C. Mallerton.

(brother of Sir H. R.)
MOH of Rutland

INSTITUTE OF NEUROLOGY



The
ROCKEFELLER
MEDICAL LIBRARY

The Library of
The National Hospital for
Diseases of the Nervous System
Queen Square.

Given by Dr. R. T. C. Pratt.

Ac. No. 1792 1,001.

CLINICAL LECTURES

ON

PARALYSIS,

CERTAIN DISEASES OF THE BRAIN,

AND

Other Affections of the Nervous System.

"Multum egerunt, qui ante nos fuerunt, sed non peregerunt; multum adhuc restat operis, multumque restabit, nec ulli nato post mille sæcula præcluditur occasio aliquid adhuc adjiciendi."—SENECA.

CLINICAL LECTURES

ON

PARALYSIS,

CERTAIN

DISEASES OF THE BRAIN,

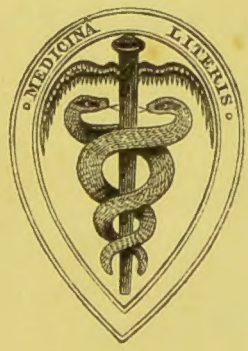
And other Affections of

THE NERVOUS SYSTEM.

BY

ROBERT BENTLEY TODD, M.D. F.R.S.

PHYSICIAN TO KING'S COLLEGE HOSPITAL.



Second Edition,

WITH CORRECTIONS AND ADDITIONS.

LONDON:

JOHN CHURCHILL, NEW BURLINGTON STREET.

MDCCCLVI.

ROCKEFELLER INSTITUTE
CLASS HIST. N.
ACCN. 1,001.
SOURCE
DATE

TO

WILLIAM STREET, ESQ.

OF NORWOOD, SURREY,

This Volume is Dedicated,

IN ACKNOWLEDGMENT OF A LONG AND VALUED FRIENDSHIP.

Feb. 1854.

ADVERTISEMENT.

These Lectures were delivered in the Theatre of King's College Hospital, on various occasions, during the last ten years. Influenced mainly by the expressed desire of a large number of those, to whom they were addressed, I am now induced to collect them.

Having been given from time to time, as cases presented themselves, which demanded explanation, or afforded illustration, they must not be regarded as forming, or intended to form, a systematic course, or even part of one.

The present series treats of certain affections of the nervous system. They have been printed from the MS. notes of one of my pupils, which were subsequently revised by myself.* Part has

* To Dr. Hyde Salter, Dr. Lionel Beale, and Mr. (now Dr.) Conway Evans, my best thanks are due, for the assistance, which they have rendered me, in the lectures of this volume.

already appeared in one of the weekly medical periodicals; the remainder is now published for the first time. All have been subjected to fresh revision, and I have added more cases, and some details not suited to oral discourses.

Should it appear, that I have not over-estimated the fitness of these lectures for publication, (and I am not without misgivings on this point,) I shall venture to furnish some further contributions of a similar nature, not only on nervous but on other diseases likewise.

26, *Brook Street, Grosvenor Square,*
February, 1854.

ADVERTISEMENT

TO THE SECOND EDITION.

A Second Edition of this book having been called for, the Author has taken the opportunity of carefully revising the Lectures, and making some additions and alterations.

The object of the Lectures has been to teach, by examples, various important points in the natural history, pathology, and therapeutics of paralytic, cerebral, and other nervous affections. The Author has seen no reason to alter or modify any of the views, whether of pathology or therapeutics, expressed in the first edition; and he now ventures again to submit his opinions, after due reconsideration, to the friendly criticism and impartial judgment of his professional brethren.

26, *Brook Street, Grosvenor Square,*
August, 1856.

CONTENTS.

LECTURE I.

PAGE

General observations on paralysis—It is a symptom, not the essential disease—Causes which may give rise to paralysis—The centre of volition—Case of paralysis from the poison of lead—Symptoms of lead-poisoning—Pathology of lead-palsy—Cases of paralysis from pressure on a nerve—Treatment by galvanism—Case of hysterical hemiplegia—Peculiar characters of this form of paralysis—Treatment 1

LECTURE II.

On cases of paralysis dependent on lesion of the brain—Case of Hardwick—Pain in the head a prominent symptom—Paralysis of right arm and leg—Contrast of the leg movement in this case and in hysterical hemiplegia—Rigidity of the paralysed limbs—Affection of the face and eyeballs—Apparent exception to the rule of deviation of the tongue to the paralysed side—How explained—The diagnosis—Remarks on the parts of the brain, by lesion of which paralysis may be produced—Four different conditions of the muscles of the paralysed limbs—Their excitability to the galvanic stimulus—Treatment of the case—Case of Williams—Sudden paralysis without stertor or loss of consciousness—Disease of the heart—Diagnosis—Treatment—Effects of trials of electricity in both cases . . 23

LECTURE III.

PAGE

Concluding history of the cases detailed in Lecture II.—Difficulty of determining the exact locality of cerebral lesions—Importance of examining into the causes of erroneous diagnosis—Grounds of the diagnosis in the first case—Points in which the diagnosis corresponded with the post-mortem appearances, and in which it differed from them—Chemical analysis of an indurated portion of the brain—Post-mortem examination of the second case—Relative importance of intra-ventricular effusion, and of increased sub-arachnoid fluid 42

LECTURE IV.

On paralysis of portio dura—Importance of making an exact diagnosis of facial palsies—Case—Leading characters of paralysis of the portio dura—This nerve rarely affected by cerebral lesion—The depression of the upper lid in complete palsy of the orbicularis palpebrarum muscle accounted for—Sir Charles Bell's view of the upward movement of the eyeball in sleep disproved—Observations on this point in patients under chloroform (note)—Paralysis of the buccinator—Proofs that the paralysed nerve is the portio dura—Double supply of the buccinator muscle—Deviation of the uvula—Romberg's view of this symptom—Causes of this palsy—Rheumatic affection of the nerve—Cases—Case of rheumatic paralysis of the deltoid (note)—This palsy occurs from exhaustion—Cases—Prognosis—Treatment—Additional cases . 56

LECTURE V.

Disease of the brain—Comparison between the diagnosis of brain disease and of heart disease—Case—Hemiplegia of the right side—Diagnosis—White softening, alone or as a precursor of apoplexy—Red softening—Atrophic and inflammatory softening—Termination of the case and post-mortem examination—Influence of galvanism—Treatment of cases of this kind 90

LECTURE VI.

PAGE

Case illustrative of the production of white softening, and of apoplexy—Three sudden seizures with paralysis of the right side—Symptoms of diseased kidney—Fourth fit with paralysis of left side—Nature of the attacks—Lesion—Diseased state of kidneys, heart, and arteries—General retrospect of the case—Effects of deposits in the small arteries upon the circulation—Is there undue determination of blood to the brain in these cases?—On the practice of bleeding in the general treatment of apoplexy 105

LECTURE VII.

Three cases of diseased brain—Case of Thurston—Epileptic seizure—Second attack—Coma—Stertor—Succession of fits of convulsion—Diagnosis—Post-mortem examination—Fatty disease of the minute vessels—Its pathology—How these diseased vessels develop softening and apoplexy—Case of H. B.—Hemiplegia—Arcus senilis of the cornea—Condition of the radial artery—Diagnosis—Case of Winning—Hemiplegia with relaxed muscles—Diagnosis and prognosis 121

LECTURE VIII.

Cerebral symptoms from renal disease—Case of Jeffreys—Peculiar complexion—Diuresis—Diagnosis of contracted kidney—Prognosis—Sudden effusion of blood into the brain—Post-mortem examination—Nature and treatment of the case—Case of Beasley—Delirium—Danger of moving delirious patients 143

LECTURE IX.

On hemiplegia—Description of this form of paralysis—Example—Complete or incomplete—Phenomena—The face-palsy—The tongue-palsy—Clinical varieties of hemiplegia—Affection of sensibility in hemiplegia—Mode

PAGE

of estimating it—Arrangement according to the state of the muscles of the paralysed limbs—Influence of diseased heart and arteries in producing hemiplegia—Observations of Kirkes and Virchow—Simple hemiplegia from brain-lesions without coma and with relaxed muscles—Cases—How recovery may take place . . . 162

LECTURE X.

Hemiplegia with relaxed muscles—Cases in young subjects—Importance of attending to the state of muscles in hemiplegic cases—Why is the paralysis sudden?—Second variety of hemiplegia, with relaxed muscles and with coma—Case of Harvey—Case of Power—Of Ware—Of Shea—Termination of the hemiplegia with relaxed muscles in four ways—Illustrations—Pathology of this affection—Treatment 191

LECTURE XI.

Hemiplegia with rigid muscles: two forms—Hemiplegia with early rigidity: two varieties of this—Cases of the first variety, in which the rigidity affects only one or two muscles—Second variety, in which the rigidity is considerable, and affects several muscles—Occurs from irritation and inflammation—Case in which it was produced by arachnitis—Case from induration and irritative disease of the brain—Cases by Rostan, Lallemand, and others—Can a paralysing lesion be also irritative?—A convulsive condition of the paralytic limbs often a concomitant of irritative disease—Examples—It occurs after injuries of the head—A less severe kind occurs in connexion with softening—Cases—The rigidity greater in the upper than in the lower extremity 217

LECTURE XII.

Prognosis in hemiplegia with early rigidity—Hemiplegia with late rigidity—Cases—Cases from Andral and Romberg—State of the muscles 241

LECTURE XIII.

PAGE

Peripheral hemiplegia—Its clinical phenomena and history—Cases—Pathology of it—Dr. Cheyne's account of creeping palsy (note)—Hysterical hemiplegia—Remarks on hysterical palsy and on aphonia, as a typical instance of it—Cases—Examples of hemiplegia from organic disease in young women, to contrast with the hysterical; one recovered, the other fatal—Paralysis excited by strong mental emotion—Emotional hemiplegia—Case . . . 256

LECTURE XIV.

Epileptic hemiplegia—Its clinical characters—Cases—Nature of the paralysing cause—Cases to illustrate the state of brain which follows such epileptic paroxysms—A general atrophic state more on one side than the other—Remarks on sub-arachnoid and on intra-ventricular effusions 284

LECTURE XV.

Additional remarks on epileptic hemiplegia—Patients suffering from an enfeebled condition of the heart liable to sudden coma—Dr. Stokes' remarks and cases—Dr. Fleming's case—Epileptic seizures in the night connected with heart disease—Choreic hemiplegia—Case—Liable to be confounded with hemiplegia from tubercular disease of the brain—Condition of the nerves and nervous centres on which choreic hemiplegia depends—Importance of ascertaining the specific gravity of different parts of the brain in this affection—Dr. Aitken's case—Dr. Bucknill's and Dr. Sankey's observations—Remarks on the differential diagnosis, the prognosis, and the treatment of the different forms of hemiplegia—Case of epileptic hemiplegia (note, p. 313)—Influence of strychnia—Spinal hemiplegia—Case—Dr. Bright's and Dr. Lee's cases 308

ADDENDUM TO LECTURE XV.

PAGE

Case of temporary spinal hemiplegia 342

LECTURE XVI.

Case of epileptic coma and lead-palsy—Urea found in the serum—Characters of the lead-palsy—Beneficial effects of elaterium—Cause of the palsy—How the lead was introduced into the system—Termination of the case—Post-mortem examination—Determination of the specific gravity of various parts of the brain—Microscopic and chemical examination of the paralysed muscles—Case of epileptic coma and gouty kidney—Examination of the brain—Specific gravity of parts of it—Chemical search for urea—Frerich's view as to carbonate of ammonia (note) 350

LECTURE XVII.

Case of syphilitic disease of the dura mater and periostitis—Remarkable effect of iodide of potassium—Diagnosis—State of the dura mater—Case in illustration—Another case—Recovery—Case with destruction of the function of the fifth nerve on the left side—Cause of the epileptic fits in these cases—Epileptiform convulsions—The value of iodide of potassium as a remedy—its effects not permanent—Additional treatment necessary in these cases—Sequel of the case of Beglin—Dr. Kennion's case of non-syphilitic disease cured by iodide of potassium—Additional case (note) 380

LECTURE XVIII.

On a case of acute idiopathic trismus—Early symptoms confined to the muscles of the lower jaw—Treatment by injections of sulphate of quinine and by chloroform inhalation—Post-mortem examination—Microscopical examination of the masseter muscles—Tetanus and trismus

PAGE

—Their difference—Distinction between acute and chronic tetanus—Death by exhaustion—Pathology of the disease—State of the nervous centres—Tetanus may be produced through the blood—Part of the nervous centres affected—Exalted polarity of them—Treatment—Use of galvanism—Cold to the spine 406

LECTURE XIX.

On cases of chorea—First case connected with rheumatic fever—Sudden supervention of chorea—Deglutition easily affected: articulation also—Mode of protrusion of the tongue—Movements chiefly on one side—State of heart—Tendency in family to rheumatic complaints—Second case—Previous rheumatic fever—One-sided—Caused by fright—Anæmic bellows-sound—Characters of the urine—Third case—Effect of attempting to restrain the movements—Treatment—Fourth case—Disposition of the malady to recur—Movements strictly limited to left side—Urine—Remarks on the pathology, treatment, and prognosis 428

LECTURE XX.

On local hysteria and catalepsy—Case of hysterical pain in the right hypogastrium—Grounds of the diagnosis—Leucorrhœa—Uterine disturbance—Forms of local hysteria—Irritable uterus—Pain in the side—Gastrodynia—Pain in the spine—Affections of joints—Differential diagnosis of true neuralgia from hysterical—Treatment of the case—Case of catalepsy 448

LIST OF CASES.

CASE		PAGE
I.	Paralysis from the poison of lead	5
II.	Paralysis from pressure on a nerve	14
III.	Hysterical paralysis	17
IV.	Hemiplegic paralysis with rigid muscles—disease of the brain	23
V.	Hemiplegic paralysis with relaxed muscles—white softening of the brain	35
VI.	Paralysis of the portio dura	57
VII.	Ditto ditto (note)	61
VIII.	Ditto ditto (spasmodic)	68
IX.	Ditto ditto (rheumatic)	71
X.	Ditto ditto (ditto)	71
XI.	Rheumatic paralysis of the circumflex nerve and deltoid muscle (note)	72
XII.	Paralysis of portio dura from exhaustion	73
XIII.	Ditto ditto	74
XIV.	Paralysis of portio dura	79
XV.	Ditto from otitis	80
XVI.	Paralysis of portio dura (rheumatic)	82
XVII.	Ditto ditto on both sides of the face	83
XVIII.	Paralysis of portio dura, of third nerve, and of part of the fifth	86
XIX.	Disease of the brain (white softening)	91
XX.	Ditto ditto, causing double apoplexy—diseased kidneys	105
XXI.	Disease of the brain, causing apoplexy	121
XXII.	Ditto ditto, causing hemiplegia, with slight rigidity	133
XXIII.	Disease of the brain, causing hemiplegia, with slight rigidity	138

CASE		PAGE
XXIV.	Disease of the brain, associated with renal disease	143
XXV.	Fatal case of delirium, with strabismus	155
XXVI.	Simple hemiplegia—recovery	179
XXVII.	Ditto ditto —recovery—death in a subsequent attack	183
XXVIII.	Simple hemiplegia—recovery	187
XXIX.	Hemiplegia, with all the muscles relaxed except the biceps—death	197
XXX.	Hemiplegia, with all the muscles relaxed—death	199
XXXI.	Hemiplegia, as Case XXIX.—recovery	201
XXXII.	Hemiplegia, with early rigidity of some of the paralysed muscles—death	220
XXXIII.	Hemiplegia, with early rigidity of some of the paralysed muscles—death	222
XXXIV.	Hemiplegia, with early rigidity of all the paralysed muscles—fatal	226
XXXV.	Hemiplegia, with early rigidity of all the paralysed muscles	228
XXXVI.	Hemiplegia, with late rigidity—death	245
XXXVII.	Ditto ditto	246
XXXVIII.	Ditto ditto —death	248
XXXIX.	Ditto ditto (Andral)—death	251
XL.	Ditto ditto (Romberg)—death	252
XLI.	Peripheral hemiplegia	259
XLII.	Ditto ditto	260
XLIII.	Ditto ditto	262
XLIV.	Hysterical hemiplegia	267
XLV.	Ditto ditto affecting one leg	269
XLVI.	Ditto ditto	270
XLVII.	Ditto ditto	271
XLVIII.	Ditto ditto of very long duration	272
XLIX.	Hemiplegia from diseased brain (syphilitic?), with symptoms resembling the hysterical hemiplegia—recovery	273
L.	Hemiplegia from diseased brain, with symptoms resembling the hysterical hemiplegia —death	278

CASE		PAGE
LI.	Emotional hemiplegia	281
LII.	Epileptic hemiplegia	285
LIII.	Ditto ditto	286
LIV.	Ditto ditto	287
LV.	Ditto ditto	287
LVI.	Ditto ditto	288
LVII.	Ditto ditto	289
LVIII.	Ditto ditto	291
LIX.	Ditto ditto	293
LX.	Ditto ditto (syphilitic)—recovery	294
LXI.	Ditto ditto with complete anæsthesia	295
LXII.	Ditto ditto —death	301
LXIII.	Epileptic hemiplegia—death	303
LXIV.	Choreic hemiplegia—recovery	313
LXV.	Ditto ditto —recovery	314
LXVI.	Ditto ditto —recovery	315
LXVII.	Epileptic hemiplegia—recovery	321
LXVIII.	Spinal hemiplegia—death	329
LXIX.	Ditto ditto —recovery	337
LXX.	Temporary spinal hemiplegia	342
LXXI.	Epileptic coma and lead palsy—death	350
LXXII.	Epileptic coma, chronic gout, and gouty kidney —death	371
LXXIII.	Syphilitic disease of the dura mater and periostitis—recovery—death at a subsequent period	381
LXXIV.	Syphilitic disease of the dura mater—death	390
LXXV.	Ditto ditto ditto —recovery	393
LXXVI.	Disease of dura mater cured by iodide of potassium	403
LXXVII.	Acute idiopathic trismus—death	406
LXXVIII.	Chorea in connexion with a rheumatic state	428
LXXIX.	Chorea almost confined to one side	434
LXXX.	Ditto, general	436
LXXXI.	Chorea—third attack	439
LXXXII.	Local hysteria	448
LXXXIII.	Catalepsy	462

CLINICAL LECTURES,

ETC.

LECTURE I.

General observations on Paralysis—It is a symptom, not the essential disease—Causes which may give rise to Paralysis—Case of Paralysis from the poison of lead—Symptoms of lead-poisoning—pathology of lead-palsy—Case of Paralysis from pressure on a nerve—Treatment by Galvanism—Case of Hysterical Hemiplegia—Peculiar characters of this form of Paralysis—Treatment.

GENTLEMEN,—I beg to-day to call your attention to the subject of Paralysis. There are at present five cases in the hospital, which exemplify different forms of palsy; I shall, thus, be able to illustrate my observations on these diseases by reference to cases, which have been under our immediate inspection.

When I use the word Paralysis simply, you will understand that I mean the loss of the power of *motion*. There is a Paralysis of *sensation*, as well as a Paralysis of *motion*. They often occur together; but the latter generally predominates. Sometimes at the commencement of an attack

they will be conjoined, but the paralysis of sensation usually disappears speedily, leaving only the paralysis of motion. Again, either may occur without the other; that which most frequently occurs alone is paralysis of motion, and that which has the readiest power of recovery, is the paralysis of sensation.

Let me make some general observations on the conditions which give rise to and attend paralysis. I must ask you to receive my statements on these points as so many *postulates;* for it would occupy too much time to enter into the proofs, which could be adduced to demonstrate the correctness of my propositions.

In the first place, then, you must not look upon paralysis as a disease of itself: it is not a disease, but a symptom of a disease. Non-medical people, and sometimes even medical men, are apt to speak as if the palsy constituted the whole essence of the malady; but this is not the case. Paralysis is an effect due to a cause, which cause itself is not always the essential disease.

What are the causes which may give rise to paralysis? They are, either an affection of the nerve or nerves, whose power is destroyed, in some part of their course, or a morbid state of the centre in which the nerve or nerves are implanted, or with which they may be less directly connected. The nervous trunks themselves may be impaired in their nutrition, the centre being

healthy, or they may have suffered some mechanical injury from violence or pressure; thus they become either imperfect conductors of the nervous force, or altogether incapable of propagating it: or some portion of the centre of volition is the seat of a morbid process, whereby the influence of the will over certain parts is suspended; the nerves of those parts, consequently, receive no impulse from that centre, whether mental or physical; and although healthy in themselves, are incapable of taking part in voluntary acts.

Whatever interferes materially with the conducting power of nerve-fibre, or the generating power of nerve-vesicles (grey matter), will constitute a paralysing lesion. Thus, in the first place, poisoning of the nervous matter will operate in this way. Soak a portion of the nerve of a living animal in chloroform, or ether, or opium, and it will fail to propagate the nervous force as long as the influence of the poison lasts. In a similar way, the poison of lead in the living system may paralyse, by weakening the conducting or generating power of the nervous matter. Poisons formed in the living system may operate in the same way; such as retained urinary or biliary principles, or the poison of rheumatism and gout. Secondly, any morbid process which greatly impairs the natural structure of nerve-matter will paralyse. Inflammation will do this; so also will atrophy, or wasting from want of sufficient

supplies of nutrient matter, as when the flow of blood is lessened or cut off. The opposite conditions of hardening and of softening of the nervous matter become paralysing lesions for the same reason, that they greatly impair or destroy the nerve-structure. Thirdly, a solution of continuity of nerve-fibre will paralyse. Cut a nerve across, and you have immediate palsy of the parts which the nerve supplies below the section. Solution of continuity from a melting down or rupture of the fibres is, I have no doubt, the frequent cause of sudden paralysis in cases of softening, or in cases of sanguineous effusions. Fourthly, pressure on a nerve or nervous centre will paralyse. Of this we have many proofs as regards nerves; a nerve, for instance, included in a ligature, or compressed by a tumor, is paralysed thereby. A fracture of the skull with depressed bone will paralyse if the brain be sufficiently compressed; an apoplectic clot on the exterior of the brain paralyses by compression; so also a tumor in its substance. I would say that the centre of volition is of very great extent: it reaches from the corpora striata in the brain down the entire length of the anterior horns of the grey matter of the spinal cord, and includes the locus niger in the crus cerebri, and much of the vesicular matter of the mesocephale and of the medulla oblongata. Disease of any part of this centre is capable of producing paralysis; but

as the intra-cranial portion of it exercises the greatest and most extended influence in the production of voluntary movements, so disease of this portion gives rise to the most extended and complete paralysis.

Another fact which I would impress upon you is one which anatomy in a great degree demonstrates, and which pathological research confirms —that the centre of volition for either side of the body is not altogether on the same side of the body. Of the centre for the left side of the body, for instance, the intra-cranial portion is on the right side, and the intra-spinal portion on the left side, and these two portions are brought into connexion with each other through certain oblique fibres from the anterior pyramidal columns of the medulla oblongata, which cross from right to left, decussating with similar fibres proceeding from left to right, which belong to the centre of volition for the right side of the body.

Having made these introductory observations, I will now pass on to the consideration of the cases; and the first we shall take is that of a man in Sutherland ward, as affording a good example of a very serious form of paralysis, of common occurrence in the London hospitals—I mean paralysis from the poison of lead.

Case I. The patient, J. Halliday (vol. xxi. p. 120), is thirty years of age, by occupation a

house-painter, of temperate habits. It appears that he has never been obliged to desist from work on account of illness until about three years ago, when he had an attack of colic, for which he was treated in an hospital in town, and perfectly recovered. He has since had several slight attacks. Three weeks ago he first noticed that his wrists became weak, and began to drop, and that he became very nervous and irritable. About this time, or rather later, he had two paroxysms of general convulsions, fits of epilepsy, during which he suddenly fell down, lost his consciousness, and struggled violently, but he did not bite his tongue. These attacks occurred once daily on two succeeding days, came on without any warning, and each lasted about ten minutes. He has frequently had cramps in the arms and legs, but no other pain in the limbs. For some weeks past he has noticed a blue line on his gums: bowels generally confined.

I have on many occasions pointed out to you the remarkable and peculiar condition of this man's arms. When they are held out, the hands drop, from his inability to maintain them in the state of extension; nor can he, by the utmost effort, bring them into the state of extension. His power of extending the fingers is also impaired, but to a less degree. If you examine the posterior surface of the fore-arm where the extensor muscles are situate, you will find that space

rendered quite concave, from the atrophy and consequent shrinking of the muscles. The fore-arm has lost its plumpness in this region, and, by pressure, you can feel the inter-osseous membrane. These are not the only muscles affected: those of the ball of the thumb are also wasted, and the movements of the thumb are much weakened, especially those of opposition. But, in this case, the wasting of these thumb muscles has by no means gone to so great an extent as you may often find in other severe cases. The flexor muscles of the fore-arms have suffered slightly in their nutrition, and have lost much of their firmness; their power is consequently much affected; and, although the patient can bend his wrists with sufficient power, he cannot grasp with full force. The general movements of the arm are accompanied with that kind of tremulousness which so frequently accompanies enfeebled states of nutrition of the muscles. The deltoid muscles are so paralysed that the patient has no power to raise his arm or maintain it at right angles with the trunk. The lower extremities are not paralysed, but they participate in the general weakness.

In addition to the symptoms above detailed, we find in this patient that curious sign of the presence of lead in the system first pointed out by Dr. Burton—namely, the blue line on the margin of the gums, present only where the teeth or their stumps are in the alveoli, and ceasing

where a tooth is wanting. There is no indication of any special lesion of the central organs of the nervous system, although these organs cannot be regarded as sound; the digestive organs are natural, as are also those of circulation and respiration; the pulse is about 70, and feeble; and the secretions healthy.

It is not very common, in lead-palsy, to see the muscles above the elbow so much weakened as in this case; not only were the biceps and triceps thus affected, but the deltoid was so much paralysed that the man could scarcely raise his arm, much less extend it at right angles to his body. He still has, although some time under treatment, a symptom which was much more obvious at first—namely, a trembling, agitated manner, like that of an intemperate man in a state of incipient delirium tremens. It is not improbable that this, to a certain extent, arose from intemperance, for, although he did not call himself intemperate, he was fond of his glass; and intemperance is a very common vice among those of his trade. I believe, however, that it mainly depended upon a general diffusion of the lead poison through his muscular and nervous systems. Again, you will remember that he had epilepsy, and evidently in connexion with the same causes which produced paralysis; the fits were slight, but still they were distinctly epileptic; they had all the essential characters of that disease: there was the sudden

fall, the loss of consciousness, the convulsion. He has had, moreover, cramps in the arms and legs.

The question here arises—what is the particular tissue or organ affected in the paralysis of house-painters and others exposed to the influence of lead? I believe that the muscles and nerves are early affected, and that, at a subsequent period, the nervous centres become implicated. The muscles are contaminated, and their nerves participate in this contamination. The nervous system is therefore affected first, at the periphery, in the nerves; and the poisonous influence continuing, the contamination gradually advances to the centres, as is sufficiently shown by the fact that the local paralysis always precedes, often for a considerable time, the epileptic convulsions or other symptoms of centric disease. In this case the epilepsy showed itself unusually early.

Another question suggests itself to us here—what is it that thus contaminates the muscular and nervous tissues, and impairs their functions? To this we answer, without hesitation, it is lead, existing in the affected tissues. If you examine the gums of patients suffering from lead-palsy, you will perceive a blue line on, or rather in, the gum, close to the neck of each tooth; and this is caused by lead existing there, perhaps as a sulphuret; you may produce precisely the same effect by the administration of acetate of lead, just as you may excite salivation by mercury.

The most positive evidence, however, is afforded by the post-mortem examination of patients who have died from lead-poisoning; for from their muscles and brains chemists are able to extract lead in notable quantity.

In hospital practice, house-painters, whose employment leads them to use lead in large quantity, are those whom we have most frequently to treat for this malady. These men get the lead paint upon their skin, where it may become absorbed, or inhale it into their lungs in the form of small particles of the powder with which the paint is made, floating in the air;* or it may be mixed with the saliva, and so get into the stomach. From one or all of these sources the lead gets into the circulation, and during its course through the body becomes deposited in the affected organs, or combined with their constituents in some way or other.

But why, it will be asked, does it alight upon the muscular and nervous tissues chiefly? why upon the muscles of the extremities, rather than those of the trunk? and why upon the extensor muscles in preference to the flexors? The answer which appears to me most satisfactory, and which

* I have heard house-painters say that that which does them most harm is the paint called *flatting*, which is largely intermixed with turpentine, which, passing off in vapour, forms a ready medium for the inhalation of lead particles.

offers the best explanation of these phenomena, is this—that those tissues in which the nutrient changes are most active receive the largest proportional supply of blood, and that blood, being loaded by a poisonous material, would impregnate them with it to a greater degree than other tissues in which the circulation is less active;* that, for this reason, such highly-nourished structures as muscle and nerve become poisoned early; that, as the muscles of the upper extremities are used more, and probably on that account experience more active nutrient changes than those of the trunk and lower extremities, the former are

* If we suppose that the activity of nutrition is equal in all tissues, then the supply of blood to particular tissues would be regulated by the bulk of the tissue—that is to say, each tissue would receive a supply of blood exactly adjusted to its size. We know, however, that the activity of the nutrient changes varies very much in the different tissues—those of muscle and nerve being highest in the scale. But all muscles and all nerves are not equally active in their nutrition; those whose function is most active and energetic doubtless exhibit the greatest amount of nutrient change, and draw most largely on the circulating fluid. And, in like manner, it may be said of those muscles which are symmetrical, that if those of the right side are more used than those of the left, the former would attract more blood than the latter; and any poisonous matter which may exist in the blood, and may have an affinity for the muscular or nervous tissue, will be drawn to that side in greater quantity than to the other, and will therefore affect those muscles to a greater extent than their fellows of the other side.

poisoned first. Moreover, in painters, the extensor muscles of the arm, as well as the muscles constituting the ball of the thumb, become principally paralysed, because they are most exercised during the practice of painting; and as they are more exercised, are consequently more supplied with blood —poisoned blood—to repair the waste that is going on in them.

Patients who *die* of lead-poisoning are generally persons who have been exposed to its influence for a long time, or have addicted themselves to intemperate habits. The morbid appearances in the brain and spinal cord denote imperfect or depraved nutrition of those centres, and are frequently associated with marks of chronic irritation of the membranes, such as accompany intemperance; these changes may be doubtless also due to the presence of lead. The brain especially presents the appearance of an ill-nourished organ: pale, soft; its convolutions wasted; the sulci between them wide; and sometimes patches of white softening are found in the hemispheres. I have seen this condition in patients who have experienced several paroxysms of epilepsy before death, or who have died in one, and in whose brains lead has been detected.

Treatment.—In the treatment of lead-palsy, the great object is, if possible, to eliminate the poison from the body, and to prevent the introduction of further supplies of it.

The patient should be kept clean, should wash much, and use such means as friction, exercise, &c., to stimulate the excreting power of the skin.

It has been thought that sulphur, when introduced into the system, has the power of neutralizing the effects of lead, by forming some innocuous compound with it; whether or not any such compound is formed I cannot say, but I have certainly found sulphur a very useful remedy in the form of a sulphur-bath. The bath which I usually employ consists of from two to four ounces of sulphuret of potassium, mixed with from twenty to thirty gallons of water. I use this empirically; and I am quite sure the patients derive much benefit from its employment.

Iodide of Potassium, as we learn from the experiments of Melsens, promotes the elimination of lead, and may be used with advantage in these cases, and in large doses. I often give along with it the citrate of iron. Frequently patients suffering from lead contamination are much benefited by the use of iron in some form or other. Sandras recommends the use of the hydrated persulphuret of iron, made by the addition of an alkaline sulphuret to a solution of a persalt of iron.*

* Bouchardat, Annuaire de Thérapeutique, 1844, and Sandras, Maladies Nerveuses, 1851. For the formula of the syrup, see Bouchardat's Nouveau Formulaire, p. 232. Paris. 1851.

Galvanism, as a local stimulant to the nerves, should not be neglected: I am certain it is of service. Our patient Halliday was much improved by it; and to its use I mainly attribute the recovery of the power of moving his deltoid muscle, which he has now attained.

In the use of galvanism, you must take care not to continue its application too long each time. Half-an-hour each day, or still better, ten minutes or fifteen minutes at three different periods of the day, will be found quite sufficient.

Added to this, the subject of lead-palsy should breathe pure air, and have good sustaining food.

Case II. (vol. ii. p. 111.) The next case, gentlemen, is one of paralysis of the arm produced by a bandage improperly applied to a man who had suffered fracture of the clavicle; and I hope that from it you will not only learn an important lesson in pathology—namely, that pressure on a nerve is capable of producing paralysis of the parts supplied by it, and likewise the particular treatment which paralysis so produced requires; but that you will also deduce a moral from it,—that a surgeon cannot be too careful in watching cases that are under his care, and in noticing every symptom which may indicate that his patient is not progressing favourably; for had that been done in the present instance, this man would not have come to our hospital with paraly-

sis of his arm. I am happy to say that the bandage was not applied at King's College Hospital, and, indeed, from the character of the gentlemen who have filled the office of house-surgeon, I believe such an incident is not likely to have happened here.

The patient who is the subject of this case has been in the hospital before under my care, for some pectoral complaint, when a full report of his history, his former health, and habits, was taken; the notes made at his admission for his present illness are therefore brief, although sufficient for the purpose: I will read them to you.

"Timothy Sullivan, admitted into Rose ward, November 18th. This patient is twenty-three years of age; has lived in London for about a year; by occupation, a labourer. Last June he was admitted into this hospital, suffering from pain in the side, and cough; both these symptoms were relieved, and he went out. Shortly after leaving the house, his right clavicle was broken by an old wall falling upon him. He went to an hospital, and the ordinary figure-of-eight bandage was applied. After a time, the patient found that his right hand and arm were numb; and soon after this, he noticed a great loss of power of the extensors of the hand. Notwithstanding these symptoms, the bandage was allowed to remain, and both the paralysis of sensation and muscular motion have continued up to the present time.

"Nov. 19th.—At present there are numbness of the hand and arm, and entire loss of power in the extensors of the hand, which is completely flexed when the arm is raised. All the muscles of the arm have less power than natural."

This case was treated with galvanism, and the patient left the hospital better, having gained some power of the extensors, and that of the flexors being nearly restored to its healthy state. It was some time, however, before the improvement became manifest. In the reports of the 22nd and 26th of November, it is stated that no change had taken place;—he first began to mend on the 28th.

Paralysis produced by pressure on the axillary plexus of nerves is not of uncommon occurrence. I have seen some cases in which it was produced in the following way:—A man gets intoxicated, and falls asleep with his arm over the back of a chair; his sleep under the influence of his potations is so heavy, that he is not roused by any feelings of pain or uneasiness, and when at length he awakes, perhaps at the expiration of some hours, he finds the arm benumbed and paralysed. It generally happens that the sensibility is restored after a short time, but the palsy of motion continues: cases of this kind sometimes derive benefit from galvanism, but if the pressure, which caused the paralysis, has been very long continued, they seldom come to a favourable termination. Nerve-

tissue is one which never regenerates quickly and seldom completely, so that any great or long-continued lesion of its structure is not likely to be removed.

I shall next call your attention to a case illustrative of another form of paralysis—namely, hysterical paralysis.

Case III. (vol. xxi. p. 170.) The following is the report of the case:—Mary Leigh, æt. forty-two years, was admitted into Lonsdale ward on October 28th, 1847: states that she is a native of, and has resided all her lifetime in London; she lived in occupation as a housemaid for twelve years, when she was married; has been a widow for seven years; had an attack of rheumatic fever when she was fifteen years of age, and a second about nine years ago; three years since she had erysipelas in the left ankle; and twelve months back she suffered from typhus fever.

About six weeks ago she worked hard for several days in succession, during which time she suffered from headache, and going to bed tired on a Saturday night, fell asleep almost immediately. About five o'clock on the following morning, she woke up with pain and loss of power in the right arm. For this she applied to a druggist, who purged her and gave her a liniment for the arm. About three weeks after this, she became an out-

patient at this hospital: took mineral and saline purgatives for a fortnight, when the leg also became affected like the arm, with pain and loss of power; she also suffered from pain in the head, and dimness of sight.

In this case the most important points to be remembered are these:—In the first place the invasion was sudden, and occurred after hard work, and it was not accompanied by any loss of sensibility, nor impairment of intellect. The face is quite free from paralysis; and this, considering the extent of the paralysis elsewhere, is a remarkable circumstance. I was at first, however, disposed to think that there was a small amount of facial paralysis; but I am now quite sure that such is not the case, and that what I took for palsy is nothing more than that want of symmetry on the opposite sides of her countenance, which the majority of people present. Examine the faces of a number of persons collected together, as I, with a numerous class before me, have now an opportunity of doing, and I will venture to say that, without any disparagement to the good expression of the countenance, you will find but few which exhibit perfect symmetry. The tongue, too, at first sight, appeared to be paralysed; but we soon discovered that the obliquity in the direction in which the tongue was protruded was due to a cause which will be very apt to mislead you if you are not prepared for it,—namely, an

undue projection of two or more of the teeth in the lower jaw, which gave an oblique course to the movement of the tongue.

Our patient exhibits no certain sign of brain disease; all the symptoms under which she labours may have occurred independently of disease of that organ. There was no injury, no impairment of intellect; the function of deglutition was unimpaired; there was no tongue or face paralysis; pain of the head there was, but this was not fixed in its position. All this militates against the idea that these symptoms were caused by any lesion of the brain. What, then, it may be asked, did cause them? We know that there are certain conditions of the system—*hysterical*—in which organic diseases are simulated by mere functional disturbance, and that even the most grave affections are occasionally imitated with great accuracy, and among these paralysis. Hysterical paralysis, however, generally affects only one limb, or a portion of one limb, as a joint or a finger.

The case of Mary Leigh, which we have just been considering, I believe to be one of hysterical paralysis in its least common form, being far more general than usual, and nearly amounting to hemiplegia. Added to the negative evidence which I have already adduced, there is much positive evidence to show that the malady is an hysterical affection; the patient's physiognomy is hysterical, as well as her general constitution;

the catamenia have been irregular; she has had decided globus hystericus, and is in the habit of voiding large quantities of very pale urine of low specific gravity. Again, the great extent of the paralysis in the limbs, and the total absence of it in the face and tongue, are certainly evidence in favour of its hysterical character; for although hysterical paralysis occurs in all parts of the trunk and extremities, it very rarely, if ever, attacks the face. But I would particularly call your attention to the peculiar character of the movement of the paralysed leg when the patient walks, which, in my opinion, is characteristic of the hysterical affection. If you look at a person labouring under ordinary hemiplegia from some organic lesion of the brain, you will perceive that, in walking, he uses a particular gait to bring forward the palsied leg: he first throws the trunk to the opposite side, and rests its entire weight on the sound limb; and then, by an action of circumduction, he throws forward the paralysed leg, making the foot describe an arc of a circle. Our patient, however, does not walk in this way; she drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no act of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks. This I believe to be characteristic of the hysterical form of paralysis.

Were I to enter into the pathology of this case

at full length, I should have to discuss the whole subject of hysteria,* which alone would occupy more than one lecture to do it justice; I must at present content myself by stating that I believe hysterical paralysis is caused by a depraved nutrition of the nerves of the limb affected, or of some part of the centre of volition. Moral causes, no doubt, exercise an important influence in the production of this state, and the power of the will becomes impaired; but that a depraved state of general nutrition, which tells chiefly upon the nervous system, or upon parts of it, is at the foundation of the malady, I think no one can doubt who considers fairly its natural history.

In the treatment of these affections you must direct your attention chiefly to the improvement of the general constitutional state of your patient, by diet, by good air and exercise, when they can be obtained, by cold bathing and improving the condition of the skin, by the use of such vegetable or mineral tonics as may be suitable to her digestive organs, and by regulating the action of the bowels, and promoting the renal, uterine, and other excretions. Many cases are perfectly curable by these means alone; and all cases should be treated in this way at first. The attention should be diverted as much as possible from the paralysed limb or part, and the exercise of the latter promoted by indirect means.

* Vide on this subject infra Lect. xiii.

If these expedients fail, then local treatment may be had recourse to. And for this purpose galvanism is, I think, very useful: it must be employed gently, so as not to alarm the patient, and its intensity may be gradually increased and varied according to circumstances. The galvanic trough may be used at first, and afterwards the coil machine, which, however, admits of easy variations of intensity, from shocks scarcely to be felt, up to those of such intensity as to be beyond endurance. Such violent shocks you will not, of course, have recourse to; their influence extends beyond the affected parts, and is calculated to disturb the healthy action of the nervous centres. Mild shocks applied for short periods, two or three times in the day, varying the direction of the current, allowing it to pass at one time from centre to circumference (direct), and at another time from circumference to centre (inverse); this mode of applying electricity you will find most successful in restoring the healthy action of the paralysed parts.

[The sequel of the case of Mary Leigh justified the diagnosis and the treatment, which latter was limited to the administration of quinine in small doses, regulation of the bowels, good diet, and the application of galvanism. She remained in the hospital from October 28th to December 4th, on which day she was discharged, having completely recovered the power of her limbs.]

LECTURE II.

Cases of Paralysis dependent on Lesion of the Brain.

In my last lecture, gentlemen, I stated to you the principal causes capable of producing paralysis, and called your attention to three cases then in the hospital, in one of which the paralysis depended on the presence of lead in the system; in the second, it was caused by a local injury to the nerves of the upper extremity by a fractured clavicle, or rather by the treatment which was adopted in curing the fracture; and the third was one of hysterical hemiplegia. All of these have now left the hospital; the first much improved, the second but slightly so, and the third quite cured.

I purpose now to speak of some cases of paralysis which have their origin in disease of the brain. A prominent feature of this kind of paralysis is its one-sidedness, constituting that which is called *hemiplegia*, or paralysis of one side of the body from disease of the opposite half of the brain.

Case IV. (vol. xxi. p. 190.) The first case to which I shall refer is that of Thomas Hardwick,

aged forty-nine, a smith, of temperate habits. This man was first attacked eight weeks ago with pain in the region of the left parietal bone; this was followed by dimness of vision, and often double vision. These symptoms continued a month, and he then had what he calls *rigors*, affecting the right arm and leg, which were probably convulsive movements of those limbs. These, he says, "turned to erysipelas," and were followed by loss of power in this leg, and afterwards in the arm.

On his admission, Nov. 10, 1847, he was suffering from pain in the left side of the head; there were loss of power, affecting both the right arm and leg, and loss of sensation in the arm; the loss of power was greater in the arm than in the leg. In walking, he drags the right leg at the same time that he lifts it from the ground, by inclining the trunk to the opposite side.

The contrast between the movement of the paralysed leg in this case and in the case of hysterical paralysis to which I referred in my last lecture, is very striking. In this case the leg is lifted from the ground; but in the hysterical case it is dragged along as if dead, without the slightest attempt to lift it. As both patients were for a long time in the hospital together, you have had abundant opportunity of observing and contrasting the different kind of movement in each.

The paralysed limbs exhibit considerable rigidity

of the muscles; this becomes particularly obvious in the arm when an attempt is made to extend the forearm upon the arm, the biceps becoming rigid, as if it resisted extension. This resistance on the part of the biceps to the complete extension of the forearm upon the arm, is often the only mark of any irritated condition of the nerves or muscles of the palsied limb. While the limb is quiescent, the muscles are soft and relaxed; but the moment extension is attempted, the biceps becomes firm and resisting. The extending force excites the biceps by reflexion, when there is even the slightest degree of irritation in the nerves of the affected limb.

There is, also, in this case, palsy of the right side of the face, denoted by hanging of the cheek, and by paralysis of the buccinator muscle. The movements of the eyes present a very peculiar appearance: they are constantly directed downwards, with a convulsive action of the depressing muscles. When desired to open his eyes, or direct them in any way by means of a strong voluntary effort, this movement of the eyeballs becomes more excited, and is accompanied by very marked convulsive twitchings. In consequence, no doubt, of these irregular movements, vision is sometimes double; the pupils are unequal, the right being the larger. These symptoms clearly indicate some irritative disease affecting the third pair of nerves, either in their course or at their origin. You will observe

that the power over the orbicular muscle of the eyelids has not been at all impaired, indicating that the *portio dura* of the seventh pair of nerves is untouched. In general, in cases of hemiplegic paralysis, the tongue deviates to the paralysed side. This case was an apparent, though not a real, exception to this rule; for the tongue deviated to the sound side. On careful examination, however, it was found that certain projecting teeth in the lower jaw diverted the course of the tongue from its ordinary channel into a deviation to the right side; and the fact illustrates the remarks which I made on this subject in my last lecture.

Now the points, in this case, which served for the foundation of a diagnosis were, first, the existence of pain; next, the occurrence of paralysis on the opposite side to the pain; and, lastly, the irregular movements of the eyeballs, and the double vision.

The existence of fixed pain in the head in general indicates intracranial irritation. Pain in the head may be situate in the course of some of the nerves of the scalp, over the brow, or across the forehead, or in the temple, or spreading upon the parietal or occipital bone, or at the vertex. Pain in any of these situations may shift, or intermit, or it may give the sensation of a nail being driven into the head—*the clavus hystericus*. When pain exhibits such characters as these, it is

not, in general, indicative of any mischief going on within the skull, but is rather symptomatic of deranged digestion, or of some constitutional disturbance, or of a hysterical or hypochondriacal state, or it is the result of debility or exhaustion; but where the pain whether sharp and burning, or dull and heavy, is fixed in its situation, as in this case, and varies only in intensity, and not in locality, it may generally be referred to intracranial irritation, such as probably would arise from disease of the membranes, or of some superficial parts of the brain. Disease of the corpus striatum, or of the optic thalamus, does not usually produce pain which is distinctly referable to a particular spot. When disease of these parts occurs, it either causes no pain at all, or a dull, heavy pain, more or less diffused; unless, indeed, the pia mater in connexion with them be extensively diseased. If the dura mater, or the arachnoid, or the pia mater, become the seat of the disease, then pain is produced, and the patient refers it to a position, which very nearly corresponds to the site of the morbid lesion: hence such pain as our patient suffers may be looked upon as indicating rather a superficial than a deep-seated lesion.

Another important symptom under which this man laboured was dimness of vision, which also assumed the form of double vision. This symptom, although it sometimes occurs independently

of cerebral lesion, ought, nevertheless, always to excite the suspicion of such lesion, and more especially if there be at the same time any affection of the muscles of the eyeball.

The paralysis in this case is of that kind which generally depends on cerebral lesion, its one-sided character denoting a cerebral rather than a spinal origin: at the same time, you must bear in mind, as I pointed out in my last lecture, that a similar form of paralysis may take place, as the result of hysteria, where there is no appreciable lesion at all. In this case there is little reason to suspect hysterical paralysis, because the face is affected, and because the mode of moving the leg is essentially different from that of the hysterical palsy; the patient is also of the male sex, which is not very liable to these hysterical affections.

The parts of the brain, the lesion of which is most apt to produce hemiplegia, are the corpus striatum and the optic thalamus, and the most frequent lesions of them are softening, a clot, or abscess. It is remarkable that lesion of the optic thalamus should produce nearly, or precisely, the same effects as lesion of the corpus striatum. This is probably explained by the intimate union of the two bodies, so that neither can be affected without the other suffering more or less the morbid influence; but if the optic thalamus be the part diseased, the corpus striatum will suffer more in consequence than the optic thalamus

would, if the corpus striatum were the seat of lesion, because of the great size and extensive connexions of the former, and the smaller size and more limited connexions of the latter. Disease also in the immediate vicinity of these parts will cause paralysis; but if the lesion be situated quite near the surface of either hemisphere of the brain, and be not of such a nature as to produce pressure, there will be no paralysis.

A clot, or an abscess, or a tumour, in the middle of the centrum ovale, will not produce paralysis if it do not cause pressure, or interfere materially with any of the fibres of the corpus striatum.

Another condition capable of producing hemiplegia is inflammatory or other disease of the membranes of the brain. The dura mater cannot suffer long from inflammatory disease without implicating the arachnoid or pia mater. When you get inflammation of these membranes, you have effusion of lymph or of pus, which, as it increases, causes pressure on the surface of the brain, which is then extended to the corpus striatum and optic thalamus, and thence results the paralysis.

If some of the deeper-seated parts, such as the crura cerebri, are affected, we also have paralysis; because the crura cerebri, as the bond of union between the corpora striata and spinal cord, form a part of the great centre of volition. Disease of the cerebellum or its crura, provided it be deep-

seated, will also produce hemiplegia; this is probably due to the connexion which is formed between the hemispheres of the cerebellum and the fibres of the pyramids in the pons Varolii.

Now, in the case of Hardwick, the first symptoms were those of irritation, producing convulsive movements of the right side; and these were followed by incomplete paralysis of the limbs. This slow accession of the paralysis, following symptoms of irritation, gives us some clue to the nature of the exciting lesion. These phenomena are precisely such as one would expect, where the lesion consisted in inflammation of the membranes of the brain, accompanied by effusion of lymph. In the first stages of the inflammatory affection you would have irritation, and consequently convulsive movements; and in a later stage, where the lymph came to be effused, we should have pressure and paralysis; but as the pressure was not excited immediately, but only indirectly, upon the centre of volition, the paralysis would be incomplete.

A very interesting and important feature in the paralysis in this case, is the accompanying spastic or rigid state of the muscles. This rigidity, according to my experience, if it supervene *early* in the paralytic seizure, or simultaneously with the paralysis, indicates irritative disease within the cranium. It is not uncommon, however, to meet with cases in which there has been very complete paralysis, with perfect resolution of the muscles;

but after a time these muscles slowly become rigid, the fingers become flexed, and sometimes firmly pressed against the palm of the hand, the hand bent upon the forearm, and the forearm upon the arm, with a tense and spastic, although wasted, condition of the muscles. This *late* form of muscular rigidity you must carefully distinguish from the *early* one, inasmuch as the former indicates that there has been loss of substance in the brain, and that the cicatrix is undergoing contraction.*

You will meet, in practice, four different conditions of the muscles in paralytic limbs in different cases. The first differs scarcely at all from that of the healthy muscles ; the muscles exhibit, perhaps, less firmness, and are less excitable by the galvanic stimulus, when the paralysing lesion is not of an irritative kind. A second condition presents complete relaxation of the muscles: they are soft, imperfectly nourished, and waste with wonderful rapidity ; so that under a paralysis of a few days' duration the size of the limb experiences a very marked diminution. In these muscles there is very little excitability to the galvanic stimulus—sometimes almost none. This is the most complete condition of paralysis, in the strict sense of that term, and it is sometimes accompanied with phenomena which denote a depressed state of the

* Vide infra Lect. xi. and xii.

general nutrition of the limb: the pulse in the large arteries of that side is weaker; there is sometimes more or less of œdema, especially if the limb be kept in a dependent position; and the heat of the limb is imperfectly maintained. Some of these cases get well; others continue paralysed, although the general health of the patient improves, and the muscles become wasted to mere membranes; others, again, continue paralysed, but the muscles gradually assume a condition,—the third to which I wish to call your attention,—of contraction and rigidity, the flexor muscles always exhibiting this state to a greater degree than the extensors. The muscles are still wasted, but they are stretched like tense cords between their origins and insertions. The biceps in the arm, and the hamstring muscles in the thigh, project beneath the skin like tense membranes. This condition is due to a chronic shortening of the muscles themselves: they are tense, but not firm nor plump; it is undoubtedly a form of muscular atrophy, of which a contracted and rigid state is a prominent feature. A fourth condition is illustrated by our present case. The muscles suffer very little, or not at all, in their nutrition; they are either constantly firm and rigid, or become so on the slightest movement of the limb; the paralysis is seldom complete. In these cases there is more or less of an exaltation of nutrition,—the circulation in the limb is vigor-

ous, and its heat is not below the standard of the other limb; and it is frequently more excitable by galvanism than the corresponding muscles on the other side.

I must beg your particular attention to these various states in which the muscles of paralytic limbs are found. You may draw practical inferences from them of great value in treatment: when the condition of rigidity is present early, your patient will bear local bleeding or local counter-irritation, or both; and may derive benefit from these measures, provided other symptoms do not contra-indicate them. The state of complete relaxation affords no indication for antiphlogistic treatment, but on the contrary, in many of the cases in which it occurs, it should be regarded as pointing out the propriety of an opposite mode of treatment. As to that condition in which the muscles assume the contracted state gradually, and some time after the paralytic seizure, I wish much it were in my power to suggest some means of arresting it. Some slight benefit is gained by subjecting the limb to frequent extension at stated periods in the day: this, I believe, will retard the contraction, so long as it is diligently persisted in; but when it has been laid aside, the contraction will go on just as if the extension had never been employed. The case is analogous to that of stricture in the urethra, or of the cicatrix after a burn, which exhibits a remarkable tendency to

contract, requiring in the former case the long-continued use of the bougie, and in many instances its frequent employment throughout the entire life of the patient. In both instances, indeed, I believe I am correct in saying that surgeons have hitherto failed in finding any means to check effectually the tendency to contraction.

I may add that long-continued and forcible extension of the limb gives rise to considerable pain when the muscles are in a state of chronic contraction—pain so severe that the patient cannot bear extension for any length of time.

But to recur to the case of Hardwick. From the various symptoms I have detailed to you I have been led to the following diagnosis in this case—namely, that the lesion is of an inflammatory kind, and that it is principally and primarily meningeal; so far I can speak without hesitation, but in determining the precise locality more difficulty is experienced; I have no doubt, however, that it is so situated as to affect the optic and third pair of nerves; and from the position, to which the man has always referred the pain, namely, to the left parietal bone, I should assign as its locality the dura mater, and the other membranes in the vicinity of the anterior and inferior angle of that bone; thence the disease has extended perhaps along the fissure of Sylvius, and thus it has come to involve the optic and third pair of nerves. It must be obvious to you, how-

ever, that the disease might readily have set up first in the pia mater, and may have involved the origin of these nerves through some other parts of the brain, producing precisely the same train of symptoms.

The treatment adopted in this case has been chiefly counter-irritation to the scalp, by tartar emetic ointment, and the use of mercury. These remedies have produced no good result; the patient's intelligence and memory are becoming affected, and I fear that the hemispheres of the brain are getting involved, either by extension of disease or by pressure. It is not improbable that ere long we shall have the opportunity of ascertaining how far the diagnosis is correct or otherwise.*

Case V. (vol. xxii. p. 103.) The second case is that of Catherine Williams, aged, as she stated, fifty, but looking at least sixty-five: she had been long addicted to habits of intemperance. This woman stated that she had been suffering from pain in the head for four months, and also from pain in her limbs: the pain in the head was not at all fixed, it was accompanied by drowsiness. She was a thin, pale, ill-nourished woman, and looked like one who drank more than she ate.

The week before her admission she complained of severe pain and numbness in the left hand and

* Vide infra Lect. iii.

arm: this was probably of the nature of a subjective sensation, due to an affection of the nerves at their central extremity, and not at their periphery. Affections of this kind not unfrequently depend upon disease of the brain; sometimes, however, they are confined to the trunk of the nerve, and are strictly of a neuralgic character. In this case, however, the concomitant headache and drowsiness pointed to a cerebral affection.

Previous to her present attack coming on, it appears that she had been working very hard, and drinking in proportion. One morning, whilst at breakfast, she suddenly lost the use of the left arm and side of the face, and, on attempting to get up, fell to the ground: in the evening, the leg on the same side became paralysed; the paralytic seizure was not accompanied either by stertor, or by loss of consciousness. On admission (Nov. 15, 1847) two days after this seizure, we found this woman completely hemiplegic on the left side, with the most perfect resolution of all the muscles; the facial palsy was also complete, and there was deviation of the tongue to the left side. The muscles of the paralysed limbs were wasted, and there was a slightly œdematous state. On examining the heart, we found a loud systolic bellows sound, indicative of an imperfect action of the mitral valve, allowing of regurgitation through the mitral orifice; affording one of many examples of the association of cardiac with cerebral

disease. From her habits and age, it seems probable that the mitral disease is due to atheromatous deposits on or in the valve, or to shortening of the chordæ tendineæ; similar deposits will also probably be found in the arteries of other parts of the body, and in those of the brain affecting the vessels perhaps on one side more than those on the other.

In this case the paralysis seems justly referable to disease of the brain; the paralysis has all the characters of that produced by such lesion. The lesion is not meningeal, because there are no symptoms of irritation, and because the paralysis supervened suddenly, and was complete. We must look for the cause of it in the substance of the brain, and I should be led to locate it either in, or in the immediate vicinity of, the corpus striatum, or the optic thalamus, as these are the parts most frequently affected in such cases, and as lesion of both or of either of them, but especially of the corpus striatum, gives rise to the most complete paralysis.

When the attack took place, there was no stertor or loss of consciousness: this shows that the paralysing lesion, whatever it may have been, caused no pressure on the brain, nor any great shock to that organ. The lesion, therefore, did not arise probably from effused blood, but rather from some degeneration of the cerebral matter itself—such as white softening; and this is a form

of lesion which very frequently occurs in a subject so ill-nourished as our patient, whose blood is poor, many of whose arteries are undoubtedly in a diseased condition, and whose heart, from the extensive regurgitant disease of the mitral valve, is not capable of supplying the brain with its due amount of blood.

My diagnosis of this case, therefore, is, that there has been white softening of the brain, situated at the parts which I have already named; this softening has probably existed for some time without any distinct symptoms, when suddenly some of the fibres giving way, paralysis followed with equal suddenness. It is neither impossible nor unlikely that some small clots (not of sufficient size to produce pressure) may exist in the white softened substance, produced by rupture of minute vessels.

The case has been treated upon this view of its nature. Indeed, the constitutional state of the patient afforded no indication for any other mode of treatment but that which would contribute to support and uphold. There is, however, but very little hope that any mode of treatment will be permanently beneficial, the whole nutrient function of the patient seems so seriously impaired, and it appears very unlikely that her vital powers will long enable her to struggle against the distressing influence of the cerebral disease.

I shall conclude this lecture, by calling your

attention to the influence of electricity on the paralysed limbs, in each of the cases which I have narrated.

Most of you have frequently witnessed the trials with electricity made on these patients, and can bear me out in the statements I shall make. I may first, however, call to your recollection the doctrine of Dr. Marshall Hall, that when the influence of the brain upon a limb has been withdrawn, the irritability of the muscles of that limb becomes considerably augmented, and that, therefore, in hemiplegic paralysis, the muscles of the paralysed limb are more excitable by the galvanic stimulus than those of the sound limb. The results of my experiments have led me to a somewhat different conclusion from that of Dr. Hall; and I would refer you to an account of these experiments published in the thirtieth and also in the thirty-sixth volumes of the Medico-Chirurgical Transactions. If, however, I have ventured to express a difference of opinion from Dr. M. Hall, I can truly say that I have no wish to treat with disrespect any views, which he may have put forward; but I cannot shrink from stating what I believe to be the truth, even though it be at variance with previously received opinions, however eminent the authority by which they may be sanctioned.

My experiments led me to arrange cases of hemiplegic paralysis in three classes, according to

the manner in which the electrical stimulus affects the paralytic limbs. In the *first* class, to which belongs the vast majority of the cases, the paralytic limb was acted upon by electricity very slightly or not at all, and in every instance to a less degree than the sound limb. In the *second* class, no perceptible difference existed as to the effects of electricity on the two limbs: these were cases of recent paralysis, the cause of which was not of a very depressing nature. In the *third* class, the electricity produced a greater effect on the paralysed limb than on the sound limb; the difference, however, was never very great, and such cases are not numerous: in nearly all such the paralysis was accompanied by recent rigidity of the muscles.

Now, of the two cases, which we have been describing, it was found that in the man Hardwick, electricity produced more effect on the paralysed limbs than on the sound limbs; and in this case you will recollect there is muscular rigidity. After the patient had been some time in the hospital, the paralysis became more complete, and the muscles less rigid, and, in the same proportion, their excitability to the galvanic stimulus also diminished.

In the second case—the woman Williams—electricity produced scarcely any contractions in the paralysed limbs, whilst it caused distinct but somewhat feeble contractions in the sound ones;

and you will remember that we applied electricity in this case, not only by the electro-magnetic machine, but also by the simple galvanic trough, and that with each instrument the same results were obtained.

The conclusions, at which I have arrived upon this subject, are, that when the paralysed limbs exhibit an early spastic or rigid state of the muscles, as in the case of Hardwick, they will be more excitable by electricity than the sound limbs; but if the paralysis be accompanied by a state of complete resolution of the muscles, the sound limb is most excitable to the galvanic stimulus, and the paralysed limb is sometimes scarcely at all to be excited. In the latter case, the nerves of the paralytic limb are in a depressed condition: in the former they are in an irritated condition; and the different effects of electricity in the two cases will depend on the difference of cause of the paralysis. If the paralysing lesion be irritative, the paralytic limb will be more excitable by the galvanic stimulus; if, on the other hand, it be depressing, the paralytic limb will be less excitable. Thus this difference in the effect of electricity on the two limbs may serve to guide us in our diagnosis; and we may conclude that the lesion is irritative or depressing, according as the paralytic limb is more or less excitable by the galvanic stimulus.

LECTURE III.

On cases of Paralysis dependent on Lesion of the Brain.

In the lecture of to-day I have to direct your attention to the concluding history of the two cases of diseased brain which I brought before you in my last lecture. Both of these cases have terminated fatally, as we had anticipated; and the opportunity is thus afforded us of comparing the diseased condition of the brain with the symptoms noticed during life.

The first of these cases was that of a man named Hardwick. You will remember that he was suffering from hemiplegia, with rigidity of the paralysed muscles; and that we treated him with galvanism, and, as is usually the case where there is recent rigidity of the paralysed muscles, these were more affected by the galvanic current than those upon the sound side—a circumstance which is due, as I think, to the exalted polarity of the nerves supplying the rigid muscles. You will not have forgotten that we derived, from the application of the galvanism, some aid to our diagnosis, and drew, from its greater influence on the paralysed than on the sound limb, the inference, that the lesion of the brain was one of an irri-

tative kind. In the diagnosis that I then gave of this case, I expressed my belief that it was one of meningeal disease primarily, and that the brain itself was secondarily affected. This patient's death was preceded by symptoms of effusion: he became comatose for a day or two previous to this event. Upon making a post-mortem examination, we found effusion into the lateral ventricles: this was evidently recent, for the brain did not appear to have been much compressed.

In making the diagnosis in this case, you will remember that I spoke with confidence respecting *the nature* of the disease, but hesitatingly as to *its locality*. The various segments of the encephalon are so closely connected with each other by commissural and other fibres, that the parts in the immediate vicinity of the diseased part sympathise with it to a very great extent—almost as much as if they were themselves diseased. Hence, it is very difficult, and sometimes impossible, to distinguish disease of the optic thalamus from disease of the corpus striatum, the intimate union of these two bodies causing a close sympathy between them. For this reason, lesion of the hemispheres, if situate close to the corpus striatum, gives rise to symptoms similar to those, which would arise from disease of that body itself; and, for the same reason, deep-seated lesion of the cerebellum causes the same symptoms as would be produced by lesion of one side of the pons Varolii. You will

not wonder, then, that it is exceedingly difficult to diagnose the exact locality of cerebral lesions. Certain broad distinctions may be made, with sufficient accuracy, by giving due attention to the general principles which physiology points out as to the functions of the great subdivisions of the brain; but I look upon it as impossible to determine the position of cerebral lesions with that minuteness and accuracy, with which we can discover the locality of lesions of other organs—the lungs, for instance.

I stated to you, that we should probably find the disease principally located in the dura mater, the arachnoid, or pia mater, near the fissure of Sylvius, and at a part corresponding to the squamous portion of the temporal bone. I also thought that the optic nerves or optic tracts, and the third nerves, would be involved in the disease, either at their origin or in some part of their course. In coming to this conclusion, I was mainly influenced, so far as regards the meningeal disease, by the fixed pain, which the patient suffered about the squamous portion of the temporal bone, and just in front of the meatus auditorius externus. My first impression was, that the optic thalamus was the principal seat of the disease; but this view I afterwards gave up from observing the intensity, and the constancy of position of the pain; and although I was quite prepared to find disease of a part so nearly connected

with the optic, and the third pair of nerves, as the optic thalamus, still I did not give it that importance which I had at first deemed it worthy of, and which our post-mortem examination showed that it really deserved.

I particularly wish to call your attention, gentlemen, to this subject now, while the details of the post-mortem examination are fresh upon your memories; and the more so because the diagnosis does not appear to have been quite exact. It is a duty we owe ourselves to scrutinise particularly any errors we commit, in either diagnosis or practice. Depend upon it, if you do this faithfully, you will derive great benefit from it: your experience will be infinitely more profitable than if you slur over your mistakes, without explanation or inquiry. On this account, I make it a rule never to pass by any mistake made here in diagnosis or practice; and I feel that in commenting upon such to you, I am far more likely to benefit both you and myself, than were I to dilate at length upon successful cases. The successful cases speak for themselves; the failures we would fain throw a veil over; but be assured, in so doing, we neither benefit science nor do we confirm our own principles of truth and honesty.

A diagnosis may be erroneous in two ways: the one, in which it is altogether wrong; the other, in which, although the principles upon which the diagnosis is framed may have been sound, and

have not been violated, the details may not be correctly defined. The first of these is likely to happen when our examination of the patient's condition has not been sufficiently careful; and when we have neglected to investigate his symptoms with all that accurate scrutiny, which alone can enable us to ascertain his real state; or where the information supplied to us, notwithstanding careful inquiry, has been incomplete or inaccurate. The second may occur from the absence of symptoms of a sufficiently distinctive character to give us the precise information we require, or where the attention has been unduly occupied by the inordinate development of some particular symptom: I say that, under such circumstances, although our diagnosis may have been conducted upon perfectly sound principles, it may still be erroneous in detail. Now I must tell you that, in this case, the diagnosis has been perfectly correct in principle, but erroneous in some of the details; and I think the error has been caused partly by the difficulty to which I have already alluded, of determining the precise locality of lesions in brain disease, and partly by the prominence which the pain assumed, and by its very local character, pointing to a spot not exactly corresponding with that at which the disease was situated.

I founded my diagnosis principally upon the *pain*; the *imperfect* paralysis, and the spastic state of the muscles, denoting that the paralysing lesion

was of a kind which likewise caused *nervous irritation*. Acute pain of the head is a symptom which indicates the site of the disease as either in the membranes themselves, or in some superficial part of the brain in contact with them; and it very commonly is felt at that spot in the head, which corresponds to the diseased part within the skull. Again, the imperfect character of the paralysis indicated that the morbid change was of some superficial part; for we find that the most complete paralyses are those caused by deep lesion —the nearer the lesion is to the surface, or the further it is from the corpus striatum and the crura cerebri, the less the paralysis, and *vice versâ*: and experience also shows, that an irritated state of the nerves and of the muscles of the palsied part is most frequently connected with superficial lesion of the brain, or with disease of the membranes.

I shall now detail to you the results disclosed by the post-mortem examination, and point out how far they correspond with or differ from the diagnosis formed during the life of the patient.

In the first place, we found that the disease was on the *left* side of the brain, the opposite to that on which the palsy existed,—so far, principles have not been violated; next, we found extensive *meningeal* disease, this also comporting with the inference which principles led us to draw from the persistence and the severity of the

pain in the left side of the head; thirdly, we ascertained that the disease was *inflammatory*, for the products of inflammation were distinctly developed, and, in this point likewise, the diagnosis was correct in principle.

It was wrong, however, in assigning the dura mater as being involved in the disease, for this membrane was healthy; and it was equally wrong in fixing the site of the disease at the anterior inferior angle of the parietal bone; it failed, likewise, in not having indicated that the optic thalamus was the part of the brain secondarily affected by the meningeal disease.

The membrane principally diseased was the pia mater, and that part of the arachnoid connected with it, just where, at the fissure of Bichât, the former membrane is extended into the ventricles of the brain as the velum interpositum, passing over the quadrigeminal bodies, and closely connected with the optic thalamus. Here the pia mater was much thickened by the deposition of lymph: it was extremely red, and its vessels much enlarged. It formed, indeed, quite a large, soft, vascular tumour, which must have excited a good deal of irritation in the subjacent nervous matter. In this disease of the pia mater there was quite enough to explain the severe pain in the head, and the other signs of irritation present; but it is difficult to understand the precise localization of it to a part so much anterior to the seat of disease

as the anterior inferior angle of the parietal bone; and this circumstance no doubt contributed very much to lead us astray. The optic thalamus on the diseased side appeared to be double its natural size, and by its great bulk compressed the crus cerebri of that side, which became flattened out by the pressure, and obliterated the locus perforatus. The crus cerebri of the right side must likewise have suffered some compression. This extensive compression necessarily affected the third pair of nerves on both sides, but chiefly on the left; and thus we obtained a satisfactory explanation of the peculiar convulsive movements of the eyeballs which this patient exhibited.

The corpus striatum was essentially healthy, but somewhat, though slightly compressed; and its function was probably weakened.

The augmentation of size in the optic thalamus in this case was more apparent than real; for, in truth, this body was in part wasted. A large quantity of new material was deposited beneath the inflamed pia mater, which added considerably to the apparent bulk of the thalamus. In cutting into this body it was found to be at one part soft and gelatinous, and at another indurated. The first portion was that in immediate connexion with the pia mater, and consisted of more or less perfectly formed pus. The second portion, which consisted of the posterior third of the optic thalamus, exhibited much change in the

proper texture of this part of the brain. At one part, quite close to its posterior extremity, there was a small cyst about the size of a pea, which contained pure pus, as determined by the microscope. Here and there we found minute spots of opaque, somewhat gritty matter, in which the microscope detected masses of phosphate of lime. Similar concretions of phosphate of lime were found in the diseased pia mater.

This indurated portion of the optic thalamus was submitted to chemical analysis, by my friend and pupil, Mr. Lionel Beale, jun.,* to whose skill and expertness as an analytic chemist I have been indebted on several occasions. He found that the indurated portions of the thalamus contained as much as 6·9 per cent. of the phosphatic salts; healthy cerebral matter containing, according to F. Simon, no more than 0·1 per cent. This remarkable retention or deposition of the phosphates in the diseased portion is no doubt connected with inflammation, and the arrest of the proper nutrient changes produced by it.

Thus the post-mortem inspection afforded us the most satisfactory evidence of the inflammatory nature of the cerebral lesion: the red and thick pia mater,—the puriform matter beneath it and upon the optic thalamus,—the cyst in this body containing pus,—the indurated portion of the

* Now Professor Beale.

thalamus: all these were signs of cerebral inflammation which even the most sceptical could not gainsay.

And it likewise proved the correctness of the diagnosis as to the cause of the paralysis. You will remember that I stated that the paralysis was caused by pressure, exerted not immediately, but indirectly, on the centre of volition. The pressure was found to be exerted on the optic thalamus, and through it on the corpus striatum and the inferior layer of the crus cerebri, both of which parts form a portion of the great centre of volition.

The inflammatory or irritative nature of the paralysing lesion corresponds in the most interesting manner with the augmented excitability of the paralysed muscles to galvanism, as we had ascertained more than once during the life of the patient. The explanation of this augmented excitability which seems to me to be the correct one, is this, not that the muscles have experienced any increase in their irritability, but that the polarity of the nerves is augmented by the propagation of irritation from the compressed and inflamed brain to that segment of the cord in which they are implanted. The nerves, in these cases, are more or less in the condition into which they are apt to be thrown by strychnine: their vital force—their polarity—is exalted, and they are excitable by the slightest stimulus.

In fine, we learn from the review of this case

that our diagnosis was sufficiently correct for all practical purposes—that, following the general principles which our present knowledge of cerebral physiology indicates, we obtained all the information we could desire for the proper treatment of the case. This treatment possibly might have been completely successful had the patient been submitted to it at a sufficiently early period before any great quantity of morbid deposit took place.

The subject of our second case, Catherine Williams, lingered on for some weeks; no improvement whatever took place in the condition of the paralytic limbs: they became extensively anasarcous, and the muscles extremely attenuated; and the patient died from sheer exhaustion.

The post-mortem inspection afforded very satisfactory proof of the correctness of the diagnosis in this case, both as to the nature and as to the locality of the lesion. The disease was in the very centre of the right corpus striatum, one-third of which must have been destroyed by it. The whole brain was shrunk; and the quantity of subarachnoid fluid was much increased. Several of the arteries at the base of the brain were studded with atheromatous spots. The convolutions were small, and the sulci between them large.

The general shrinking of the brain explained the increased quantity of the subarachnoid fluid.

As to the nature of the disease: the middle third of the corpus striatum was excavated into a small cavity, which was filled by fluid and softened brain-substance; probably, also, by half-dissolved softened clots of blood. On examining the contents of this cyst by the microscope, we could find no trace of any inflammatory product; there were some remains of nerve-tubes, and the rest consisted of an undefined granular matter, derived probably from the destruction of the vesicular matter of the corpus striatum.

I think there can be no doubt that in this case there was first simple softening (without discoloration) of the middle third of the corpus striatum; next came the solution of continuity of some of the fibres of the corpus striatum; and, possibly, at the same time, the rupture of some minute vessels, and the effusion of blood in small quantity. At this time the limbs became paralysed; and, owing to the total destruction of the brain-substance in a part so important as the corpus striatum, and the entire absence of any reparative effort, they never evinced the least sign of improvement, or of any other change, save wasting.

It would be difficult to find a more perfect example of a brain suffering from imperfect nutrition than this. No doubt the local softening was due to some defect in the local nutrition, the precise nature of which, however, we were not able to detect.

The nerves of the paralytic limbs were depressed in their vital powers: the want of their wonted stimulus, the will, suffered them to fall into decay; and very probably the morbid state of the corpus striatum exercised a depressing influence upon them. Hence their polarity was much below par; and the galvanic stimulus, which excited free action in the sound limbs, produced little or no effect on the paralysed limbs.

There is one point in which these cases present an interesting contrast with each other, to which I must allude before I conclude this lecture. The man, Hardwick, as you will remember, died comatose, and we found an undue quantity of fluid *within* the ventricles, and no subarachnoid fluid around the brain. The woman, Williams, died from exhaustion, without any symptom immediately referable to the brain. In this case, the subarachnoid fluid was abundant, but there was no fluid in the ventricles.

You will find, I think I may say invariably, that the accumulation of fluid in the ventricles, when it exceeds a certain amount, produces coma. In the adult the comatose symptoms come on earlier, and with a less amount of effusion, than in the child, from the resisting nature of the cranial wall in the former, whilst, in the latter, the still open state of the fontanelles, and of some of the sutures, allows the skull to expand, as the fluid in the ventricles increases in quantity.

On the other hand, the increase in the subarachnoid fluid is not in itself accompanied by any special symptoms. This augmentation of a fluid which naturally occupies the subarachnoid space, is due entirely to a shrinking or diminution in the bulk of the brain, from whatever cause; and its quantity bears an inverse proportion to the bulk of the brain. You find it in large quantity in the crania of persons dying anæmic, and also when the brain has been much impaired in its nutrition, so as to cause a diminution of its bulk; and even if there be a local diminution of bulk, as when one or two convolutions have shrunk, or have sunk-in from the destruction of the subjacent cerebral substance, you will find an accumulation of fluid opposite the shrunk or depressed convolutions.

LECTURE IV.

On a case of Paralysis of the Face, dependent on loss of power of the Facial Nerve (portio dura of the seventh pair.)

Gentlemen,—The case upon which I propose to comment to-day is one of not uncommon occurrence—a form of paralysis of the face dependent on the loss of power of the facial portion of the seventh pair of nerves.

As every form of palsy has a formidable appearance, and as this is particularly the case when the face is affected, and the more so in proportion to the greater distortion of the countenance, I advise you to make yourselves well acquainted with the various kinds of palsy that affect the face. The alarm, which a loss of power on one side and the consequent distortion of the features, occasion to the patients or their friends is very great—and naturally so. Paralysis is a formidable symptom; and on its first appearance it is apt to be looked upon as a sign of the break-up of the patient's constitution—an indication that his doom is sealed. It is very important that, under such circumstances, the medical attendant should display a perfect acquaintance with the real state of the

case, and be able to allay the patient's or his friend's fears, when it is possible to do so. Since in the generality of palsies, such as the patient now in the hospital suffers from, you may give with confidence, at least as regards the patient's life, a favourable prognosis; you ought to possess a thorough knowledge of the signs and the symptoms of this malady, so as to enable you to recognise it readily and with certainty whenever it comes before you.

Case VI. (vol. xxi. p. 228.)—The subject of this case is John Garrey; he is in Fisk ward, and you can scarcely fail to recognise him by the peculiar expression of his countenance; for on one side his look is most doleful and melancholy, while on the other it is very much the reverse.

He is thirty-nine years of age, and was admitted into the hospital on the 6th of January, 1848: the report in the case-book of that date goes on to say, that "he has lived in London ever since he was nine or ten years of age; is a married man, a carpenter by trade, temperate, and has always had good general health until a week ago, when, after keeping in-doors for a fortnight, in consequence of having a bad leg, he was obliged to go out in search of work, and was exposed all day to very cold weather; in the evening he had an attack of shivering, and twitching in the under lip on the right side; after supper he found his

lip drawn to the left side, but it was free from pain. About four days after this he was seized with a severe pain behind the right ear, which still continues, as do the distortion of the face, and inability to close the eye on the right side, even during sleep; he cannot whistle, and he frowns only on the left side. Upon trying to shut the eye he carries the ball upwards and inwards, but does not bring the lid down over it, excepting a very little. He protrudes the tongue in the median line."

The leading character of cases of facial palsy such as this, is the inability to close the eyelids, from paralysis of the *orbicularis palpebrarum* muscle: this is the pathognomonic sign which determines the peculiar nature of the palsy, and distinguishes it from the more serious form of facial palsy which is dependent on disease of the brain and palsy of the fifth or of the third nerve.

It is remarkable how seldom the seventh pair of nerves is affected by disease of the brain. I cannot say that I ever saw an instance of complete paralysis of the orbicular muscle of the eyelids due distinctly to uncomplicated disease of the brain; and I have only seen a few in which the power of the muscle appeared to be enfeebled from that cause. Thus we have a point favourable and consolatory to a patient afflicted with *portio dura* paralysis; namely, that the affection being seated

in that nerve need not excite the same alarm as to disease of the brain as in other cases of partial palsy, that of the third nerve, for instance. Moreover, disease of the brain would give rise to a different form of facial palsy.

You have only to examine this patient with care, and you will find that he has almost every sign which indicates that the paralysis has its seat in the portio dura nerve. He cannot close his right eyelids; in making the attempt, however, he seems not to have lost the power altogether, for the upper lid is slightly depressed; yet if you put your finger on the orbicular muscle you do not find the slightest contraction of it. How, then, is this slight depression of the upper lid produced? Watch him closely while he shuts the left eye and attempts to do the same with the right, and you will perceive that at the moment the left eye is closed, the right eyeball turns upwards and inwards to such an extent, that the cornea is nearly or wholly concealed by the upper lid, and by this upward movement of the ball the upper lid is slightly depressed. The same upward movement of the eyeball takes place on the sound side at the moment of the forcible contraction of the orbicular muscle. It is a very curious instance of an involuntary movement which cannot be controlled, accompanying a forcible action of another kind; and it has reference to the complete protection of the eyeball against those

sources of injury, which would occasion the forcible closure of the eyelids.

Sir Charles Bell, to whom we are so much indebted for our improved knowledge of the paralytic affections of the face, dwelt much on this upward movement of the eyeball. He affirmed that it took place in sleep, and that during sleep the eyeball retained this position. I doubt much the correctness of this assertion. I have had many opportunities of satisfying myself that in perfectly tranquil sleep the eyeball is directed forwards, and seems suspended in the orbit, being equipoised among its muscles. Close the eyelids slowly and gently, and the eyeball remains quiescent,—contract the orbicular muscle forcibly, and the eyeball instantly turns upwards and inwards. When the orbicular muscle is made to contract strongly as a reflex action, as when you try to push any object into the eye, the upward movement takes place. But in ordinary winking you have none of it. This movement of the eyeball, then, accompanies only forced contraction of the orbicular muscle of the eyelids.

If you will take the pains to watch persons sleeping, whenever you have the opportunity, you will find that in *sound* and *tranquil* sleep there is no indication of active contraction of the orbicular muscle: there are no wrinkles of the eyelid, and no depression of the brow, as when that muscle is in strong contraction;—if, with the

greatest care and gentleness, you raise the upper lid, you will find the eyeball directed forwards, maintained in this position by the equilibrium of its muscles. Should your attempt to raise the lid give rise to a reflex action, you will encounter a distinct resistance from the contraction of the orbicular muscle, and the eyeball will be turned upwards and inwards, more or less forcibly in proportion to the force of the reflex action. I think, therefore, we are justified in asserting, that in sound sleep the position of the eyeball is one of quiescence, that it is maintained in that position by the passive contraction of all its muscles, and that the eyelids are kept closed by the passive contraction of their orbicular muscle, and that, during sleep, there is no effort or influence of the nervous system directed upon any of these muscles. It is only when sleep is disturbed, when the mind is more or less active, as in dreaming, that you will find active contraction of the orbicular muscle of the eyelid.*

* Since this lecture was delivered, I have repeatedly examined this point in patients under the deep sleep produced by chloroform. And in every instance, reflex actions being in such cases in abeyance, the eyeball remained undisturbed when the upper eyelid was raised.—I have notes of one case (Case vii., Nov. 1840), in which the left eyeball (the palsy being on that side) was turned upwards and *outwards*, when the eyelid was raised. It is probable that this was due to a slight and transient palsy of the third nerve, or of the ciliary nerves, as there was

Our patient is unable to frown on the right side, while he does so distinctly on the left; neither can he move his scalp on the right side: the corrugator supercilii, and the frontal portion of the occipito-frontalis muscles, on that side being paralysed. The levatores alæ nasi, and the zygomatic muscles, are likewise paralysed on the right side, and therefore the right nostril is motionless, and the angle of the mouth hangs on that side. The orbicularis oris muscle is paralysed as to its right half; the patient is consequently unable to purse-up his mouth, and if you ask him to whistle, he will afford you indications of his inability to perform this, as well as other actions. In making the attempt to whistle, you may perceive that he contracts the orbicular muscle of the mouth on the left, but not at all on the right, and so he is quite unable to get his lip into the position necessary for the production of sound; and while trying to adapt his mouth for this purpose, he smiles or laughs, as is so often the case when you ask a person to whistle, and he thus affords you the opportunity of seeing

at the same time dilatation of the left pupil. The patient was a gentleman of decided gouty constitution;—the palsy was removed in a fortnight. In this case I noted a symptom which is alluded to by some continental writers—namely, a slight defect in the power of taste on the left side of the tongue. This was probably due to a coincident affection of the superficial nerves of the tongue.

how completely the action of the features is confined to the left side. The act of smiling or laughing is exaggerated on the left side, and the reason is because the left muscles have completely lost the resistance of those of the right side, which remain perfectly motionless, and which from disease have lost their tone, and have suffered much in their nutrition. For the same reason all the movements of the features which act in symmetry, and which at the same time counterbalance each other, are found to take place to an exaggerated extent on the healthy side. Hence in smiling, laughing, and speaking, the face is drawn more or less to the left side: the distortion takes place on the healthy side, the paralysed side remaining unmoved. The popular notion, in cases of this kind, is, that the disease is on the side to which the mouth is drawn. No medical man, however, can fall into this mistake if he be at all acquainted with the real condition of the patient.

Another muscle which is paralysed in this case, and in all cases of the same kind, is the buccinator. Hence the cheek hangs loose, and as the patient speaks, it flaps to and fro. This extreme looseness of the cheek is not an early symptom of this form of paralysis; it manifests itself more and more, the longer the duration of the disease, and ultimately becomes the cause of symptoms very troublesome to the patient. It interferes not only with articulation, from its looseness and the flap-

ping movement, while the patient is speaking, but with mastication likewise. The paralysed muscle allows the food to accumulate between the teeth and the jaw, and fails in its function of supplying the mill with its proper amount of material to be ground. After a little time, patients learn to remedy the defect of articulation which the paralytic condition of the buccinator muscle causes, by supporting the cheek with the hand; and a similar kind of support helps to remove the inconveniences of mastication.

Increasing flaccidity of the cheek, and especially a rapid development of that condition, is a symptom of unfavourable omen as regards the patient's prospects of complete recovery.

You will observe that all the muscles paralysed in this affection are *superficial*: they are all muscles more or less concerned in the expression of the countenance. The deep-seated muscles are not affected—these are muscles of mastication—the only muscle paralysed, which is concerned in mastication, being the buccinator, which is, however, only accessory to that function, and is as much or more a muscle of expression.

And now we come to a most important question—what is the exact nature of this disease? Is it a disease of certain muscles, or of a certain nerve or nerves, or is it an affection of the brain? Its one-sided character would denote its being a cerebral affection: it may, however, occur simul-

taneously on both sides, and I have myself seen two instances of this kind. Experience, however, as I have already told you, assures us that it very rarely indeed accompanies cerebral disease; sometimes it occurs as the result of *intracranial* disease, less frequently from lesion of the brain itself.* What, then, is its nature? Sir C. Bell clearly pointed this out long ago, and to him we are especially indebted for our knowledge of the precise nature of the disease; so much so, that some designate the disease "Bell's paralysis of the face." Not that I should recommend you to adopt this name; for I must say that I cannot regard it as a compliment to the great names of

* This remark applies to simple palsy of the portio dura, unaccompanied by any other symptom referable to brain disease, such as pain in the head, giddiness, confusion of ideas, affection of speech, delirium, or by any other form of paralysis. Dr. Watson records a very interesting case in which palsy of the portio dura was among the first symptoms; but, in this case, there were other, and very decided symptoms too, of brain mischief, and the lesion proved to be "a cancerous tumour, occupying the right hemisphere of the brain; at its under part a clot as big as a hazel-nut." Lectures, vol. i. p. 541. A careful analysis of the particulars of this case shows, I think, that this lesion operated only indirectly in the production of the palsy. The portio dura and the portio mollis were compressed and hardened by the superimposed tumour, which pressed down the intervening brain substance upon them.

our profession, to attach them to any of the numerous ills which flesh is heir to.

Sir C. Bell first pointed out the true nature of this palsy, because he was the first to unravel the intricacy of the nerves of the face. He showed that one nerve, and one nerve only, was at fault in this disease, and that it was strictly a local paralysis, due to a lesion in some part of the course of this nerve. The affected nerve is the portio dura of the seventh pair, the proper facial nerve, which supplies all the muscles paralysed in this affection, and is the only motor nerve which supplies nearly all of them. The fifth pair is not affected, because the muscles of mastication are free, and because the sensibility of the face remains intact. Sometimes the patient complains of slight pains in the face, which may probably be due to a slight affection of the filaments of the fifth.

There is, however, one muscle paralysed in this affection, which does receive a supply from the fifth—namely, the buccinator. This muscle has two motor nerves—a branch of the facial, and the long buccal nerve from the fifth: the former may be regarded as its nerve of expression; the latter as its nerve of mastication. How comes it, then, that if the first be paralysed and the muscle ceases to act in expression, it likewise ceases to act in mastication? The two nerves are distinct; and the buccal nerve is one of considerable size,

and to all appearance would seem perfectly adequate to the maintenance of a different action independent of the portio dura. It is not easy to find an explanation of this curious fact, which is equally true if the nerve first palsied be the fifth—as in cases of hemiplegia, in which the hanging of the cheek is due to paralysis of the buccal nerve, and of the buccinator muscle. The advocates of Dr. Hall's views would doubtless explain it by assigning to the facial nerve a specially spinal character, and to the fifth a cerebral. The palsy of the facial nerve would, according to these views, not only destroy the influence of the will over the muscle, but also cut off its supply of irritability. Without going into other objections fatal to this explanation, it is quite enough to state that it is inadequate to explain the complete palsy of the buccinator muscle when the fifth is the only nerve affected, as in common hemiplegia.

In some instances the velum of the palate participates in the paralysis; and when you look into the patient's throat, you find the uvula inclining away from the paralysed side, and the velum drawn to the sound side. It is probable that the portio dura exercises some influence on the muscles of the palate through the greater superficial petrosal nerve of Arnold, which arises from the knee-shaped swelling of the trunk of the portio dura in the aqueduct of Fallopius, and communicates with Meckel's ganglion, whence the palate-

muscles derive their nerves. Possibly this influence may be more direct in some cases than in others.

Romberg considers this symptom as indicating that the paralysing lesion affects the nerve in the Fallopian aqueduct. I have seen undoubted instances of disease of the aqueduct causing paralysis of the nerve in which this symptom did not exist. In my experience it is a symptom of very rare occurrence, and I incline to think it may be looked upon as a coincidence, like the partial defect of taste to which I have already alluded.*

* A curious instance of this attraction of the uvula to one side occurred not long ago (Feb. 1855—case of James Osborne, Vol. xlvi. p. 27) in the hospital, in connexion with a *spasmodic* condition of all the muscles supplied by the portio dura, as if that nerve were the seat of a continued irritation. The left side of the face was affected, and all the superficial facial muscles were in a state of spasm, including the orbicularis palpebrarum, which kept the eyelids constantly closed. There was imperfect paralysis of the left arm and leg, and the symptoms seemed to indicate a lesion paralysing to the left arm and leg, but approaching sufficiently near the point of implantation of the facial nerve of the left side to keep all its fibres in a state of irritation. There was a remarkable unsteadiness of gait, and the power of co-ordination was very much impaired, especially on the left side If he stretched out his hand to touch an object, it went zigzag, and ultimately far wide of the mark; and he could not carry a cup of tea to his mouth without spilling it. At first starting to walk it was necessary to support him, and he walked like a drunken man; in attempting to turn, he was always in

As this is a local palsy, its causes are generally strictly local. Thus a common cause of it, and especially in strumous children, is *otitis*, and the subsequent caries of the petrous portion of the temporal bone. In such cases the paralysis is generally very complete: it is caused by inflammatory or destructive disease of the nerve in the Fallopian aqueduct, and it is often associated with a discharge from the ear, and with deafness. Injury to the trunk of the nerve may give rise to this form of palsy: hence it often follows surgical operations on the face, and accidental wounds in the parotid region; and formerly, before the true function of the facial nerve was known, when surgeons used to divide this nerve for *tic douloureux*, this form of paralysis used to be regularly manufactured by chirurgical skill.

A very common cause of this palsy is the influence of cold; as by exposure at an open window

great danger of falling. There was slight failure of sensibility of the *right* side. The patient improved under treatment, left the hospital with more power in his left side, and better able to balance himself, but with the spasmodic state of the facial muscles unchanged, and the uvula still inclining. He has not been heard of since.

I may remark here that there is an *acute* affection of the portio dura nerve which induces most painful spasms of the facial muscles. This is essentially different from the affection in the case referred to. It occurs in connexion with gouty or rheumatic conditions, and is analogous to the acute affections of the sciatic and median nerves.

in a coach or railway carriage to a current of cold air. The case under our consideration was one of this description, the patient having been exposed the whole day to a cold atmosphere while in search of work. These are instances of what has been called "*peripheral paralysis*,"—cold acting directly on the peripheral ramifications of the nerve.

Sometimes you meet with cases which cannot be satisfactorily traced to exposure to cold; the patients, however, will be found to be out of health, and to have had pains about the face and neck for some days. It is probable that in all cases which have not a traumatic origin, or are not caused by disease of the petrous bone, there may be some constitutional fault which may show itself in this local malady, just as painful affections of sentient nerves—the fifth, for instance—are undoubtedly generally of constitutional origin.

Periodical neuralgic affections are, I believe, very frequently due to the determination of some poison to a particular nerve—as the paludal poison, or some matter generated in the system, gouty or rheumatic. There is no reason why such morbid matters should not affect a motor nerve as they affect a sensitive nerve, causing paralysis in the one case, and neuralgia in the other.

Mr. Bowman tells me he has met with several cases of distinctly rheumatic paralysis of the portio

dura, and also of some of the nerves of the orbit, among the patients at the Ophthalmic Hospital, Moorfields.

Case IX. Very lately the following case has come before me, being one of palsy of the portio dura, which had marked evidence of being rheumatic in its origin. A man, æt. 28, had severe rheumatism of some of the intercostal muscles of the left side. This got well, and then the muscles of the hip became affected, and he was completely lamed in consequence. As these were getting better, he found his face to become suddenly paralysed on one side, with all the symptoms of palsy of the portio dura. During the first few days of his illness, the urine deposited lithic acid and lithates freely. The case ended favourably.

Case X. In November, 1843, I attended, with Mr. Wetherfield, a gentleman, living in Regent-street, 85 years of age, in whom complete and well-marked palsy of the portio dura came on under the following circumstances. He had in general enjoyed excellent health, but began to suffer from pains in the right arm and right side of the face, which he regarded as rheumatic, and which did not seem referable to any other cause. These continued for several days uninfluenced by treatment, when one morning, as he was proceeding to shave, he found the right cheek hanging loose, and the face drawn to the left side. I saw

him soon after, and found all the signs of palsy of the portio dura unusually well marked. The cheek became flaccid and wasted with remarkable rapidity, and our patient suffered great inconvenience from it both in mastication and in speaking. I never saw more striking deformity than this patient presented within three weeks of the invasion of the palsy, from the staring eye and the flaccid and hanging cheek. He never evinced the slightest effort at recovery, and died within three months of senile gangrene of the toes, but without any fresh symptom referable to the nervous system.*

* Other forms of local paralysis may occur in states of constitution, if not rheumatic, at least allied to it, with imperfect action of the kidneys.

The following affords a good example of this :—

Case XI. A medical man, æt. 53, extensively engaged in practice in the county of Berks, applied to me in Aug. 1847, with complete paralysis of the deltoid muscle. He was a stout, full man, tall, of large build, and very active in his habits ; fed well, and drank beer, but not to excess. He had been subject to a shifting neuralgia of the scalp, and to a discharge from the right ear, where he thought the tympanic membrane was destroyed ; he was deaf on that side. Six weeks before he came to me he suffered from pain in the left side of the neck and shoulders, followed by complete paralysis of the left deltoid muscle and weakness of the whole arm. On examining, I found a total inability to raise the left arm to a right angle with the trunk, or to perform any of those actions which are usually effected by the deltoid muscle, which was very

This form of palsy comes from exhaustion, as in that exhausted state of system which occurs after parturition and from nursing. The following is a good example.

Case XII. A married lady, twenty-five years of age, of highly nervous temperament, was brought to me by Dr. Westmacott in May, 1851.

much wasted. He could, however, grasp perfectly with the left hand, and execute all the other movements of the arm and of the forearm. There was some degree of numbness of the arm. There were no symptoms distinctly referable to the head. His tongue was coated; appetite good; the discharge from the ear had ceased. The urine was pale, of low specific gravity, and contained albumen in small quantity.

I viewed the case as one of local palsy, connected with a deranged state of system, rheumatic or gouty. I regulated his diet, and gave him small doses of the mineral acids. After a fortnight of this treatment, he improved considerably; and could raise his arm slightly. The albumen in the urine had much diminished; and crystals of lithic acid were precipitated. He was now ordered three grains of iodide of potassium, with ten minims of liquor potassæ, thrice a day. He only followed this treatment for ten days, as the iodide of potassium purged him. Still he was improving. I continued the liquor potassæ, and advised galvanism to the muscle. This plan was diligently pursued for a fortnight, at the end of which time he had so far improved that he could raise his arm nearly to a right angle, he could put on his coat, and tie his cravat; and in three weeks more he was quite well. All signs of albumen had disappeared from his urine.

On the 5th of that month she had a miscarriage, which was followed by considerable hæmorrhage, with debility and a hysterical state. On the 13th she had inflammation, with slight ulceration of the right tonsil, which yielded readily to treatment. On the 19th, having been more than usually hysterical, after a fit of laughter, she found her face drawn to the left side, and on the following day there were unequivocal signs of paralysis of the portio dura on the right side. There was slight tenderness of the parotid region on the right side. The only treatment adopted in this case consisted in the use of warm fomentations over the right parotid region and the exhibition of the tincture of the sesqui-chloride of iron. In three weeks she was quite well.

Case XIII. As an example of this affection coming on under exhausting influence, I may refer to the case of Mr. H., æt. thirty-five, an overworked medical practitioner in a very populous district, largely employed in midwifery practice. After an unusual amount of night-work (March, 1848), he found himself paralysed on the left side of his face. All the signs of palsy of the portio dura were present. The attack was not preceded by headache, nor by any pain in the face or neck, nor any affection of the ear. This patient speedily recovered on going to the country, and taking tonic medicines.

The cause of the palsy, in our patient Garrey, appears to have been the direct influence of cold. This view is confirmed by the pain which the patient suffered at first in the neighbourhood of the ear; as if the ear itself and the nerves about it were chilled, and some degree of inflammation excited in them in consequence.

The duration of this palsy varies considerably: it rarely, if ever, lasts a shorter time than ten days, whilst it very often extends to as many weeks: perhaps three or four weeks may be assigned as an average duration for the non-traumatic cases.

The prognosis in cases of this kind should always be founded upon the cause. When the paralysis has been caused by mechanical injury, your prognosis must generally be unfavourable, more especially if any distinct solution of continuity have taken place in the nerve. Nerve-substance is very slow of regeneration; and when it is reproduced, the new fibres do not adapt themselves with precision to the old ones, and so they form very imperfect conductors of the nervous force. But if the paralysis is due to cold or to some constitutional cause, it almost invariably gets well. But you should bear in mind, that even in cases which are incurable by reason of the solution of continuity of the nerve, there is little in this form of paralysis tending to shorten life, or calculated to prove otherwise than inconvenient,

by causing imperfection of speech, mastication, and vision, and sometimes of deglutition.

I must, however, beg of you not to lose sight of the fact, that sometimes paralysis of the portio dura may be the forerunner of much more serious disease. The disease of the temporal bone, on which the palsy depends, may pass on to caries, and may excite meningeal inflammation and even abscess of the brain. A very striking example of this you will find recorded in Dr. Graves' Clinical Medicine: vol. ii. p. 569.

In Garrey's case I have given a favourable prognosis, believing that no serious mischief has been done to the nerve, and that it has not been the subject of destructive disease. He begins to gain some power over the orbicular muscle of the eyelids, and the distortion of the face is somewhat less. The duration of the palsy has already been quite three weeks, and it seems probable that the patient's recovery will not be rapid, as so little amendment has as yet shown itself. I have observed that when the symptoms begin to mend early—that is, within a week—complete recovery takes place very rapidly; but if the first signs of improvement show themselves late, the recovery is slow, or only partial.

You will likewise find it necessary to be guided by the cause of the palsy as to the course you will pursue in its treatment. If otitis be its cause, and the inflammation be of recent occurrence, it

may be necessary for you to have recourse to the usual antiphlogistic measures for its suppression; and in such a case it may be desirable to carry the use of mercury to ptyalism. In the palsy from division of the nerve, all medical treatment is useless; and when the disease has been caused by cold, or has arisen from any constitutional cause, much medical interference is not requisite. If there be pain of the face, warm fomentations will prove useful. Sometimes a few leeches at the angle of the jaw, or over the parotid space, or behind the ear, may be tried, or a blister, or iodine paint. I cannot say that I have ever known clear and distinct benefit produced by any of these latter remedies in shortening the duration of the palsy.

In the use of internal remedies you must be guided by the diathesis, and the existing condition of your patient. Mild purgatives are generally useful, and sometimes alkalies and sudorifics, and I have seen decided benefit from the use of the iodide of potassium. In the cases which follow exhaustion, tonics are clearly indicated. I cannot name to you any remedy which will act specifically on the palsied nerve. Strychnine is of no use in such cases.

As to local remedies, I advise you to abstain from the use of them, if possible. Blisters are open to this objection, that they sometimes cause enlargement of the neighbouring glands of the

neck, which, by their pressure, may increase the evil we wish to remove. Galvanism, employed carefully, may be useful,—always remembering, in the application of it, to vary the direction of the current, and never to continue it so long as to exhaust any small amount of nervous force which the nerve may be capable of maintaining.

Our patient, Garrey, has been treated chiefly by leeching and fomenting, and purging, in the first instance, and afterwards by the iodide of potassium. He has been completely relieved of pain, and his muscular power is beginning to return. I propose shortly to try the effects of galvanism with him.

Garrey suffers from a very troublesome symptom of frequent occurrence in these cases, and which is very difficult to deal with—I mean irritation of the conjunctiva, occasioning free lacrymation and soreness of the eye. This is obviously due to the constant exposure of the eye, occasioned by the loss of the power of winking; and it can only be obviated by attention on the part of the patient to the protection of the eye, or by his wearing a shade to cover it.

[This patient got quite well, galvanism having been applied for two or three days. Four weeks elapsed from the occurrence of the palsy to his complete cure.]

The following cases are of sufficient interest to

induce me to subjoin a brief detail of them in further illustration of the clinical history of this form of paralysis.

Case XIV. (vol. xvi. p. 96.) F. McCasey, æt. 28, a silversmith, of very dissipated habits, had syphilis ten years ago, health otherwise good. On the night of the 6th Dec. he was out till a very late hour, and next day observed a redness of the conjunctiva of the right eye, which did not subside readily. There was no accompanying sore throat nor any glandular enlargement. On the morning of the 13th, on getting out of bed, he found himself unable to spit. He went to his workshop, and there the peculiar condition of his features excited the risibility of his fellow-workmen, which induced him to come to the hospital to have the peculiarity investigated. All the usual signs of paralysis of the portio dura were present,—the right eye stared, and the right cheek was flaccid, —mastication was impaired on the right side, but the masseter and temporal muscles acted perfectly. The conjunctiva of the right eye was red and irritable, and tears flowed freely and to a troublesome extent. The right parotid gland was slightly painful to pressure; and he stated that since he had been salivated, six months ago, the parotid glands were liable to become tender and swollen upon the accession of the slightest cold. There was slight deafness of the right ear.

This patient was treated by the application of four leeches over the right parotid, and iodide of potassium was exhibited in the dose of three grains thrice a day. The latter was omitted on the eighth day. Under this plan the paralysis gradually subsided, and he left the hospital, cured, on the 31st of December, eighteen days after the attack.

Case XV. (vol. xxiii. p. 46.) Harriet Winter, æt. 18, a servant, previous health good, excepting that she has for the last two years suffered occasionally from pains in the head. On the morning of the 11th Feb. she awoke with severe pain across the forehead and top of the head, and numbness of the left side of the face, which was much swollen from inflammation of the gum of a carious tooth.

At the same time she was seized with severe pain as of otitis in the right ear, and the day after (Feb. 12) the face became drawn to the left side. She has also been much troubled with pain and numbness of the upper and lower extremities on the left side. She was admitted into the hospital on the 28th Feb. with well-marked symptoms of palsy of the portio dura of the right side. "She frowns, laughs, and talks with the left side only, —cannot close the right eyelid,—there is tenderness on pressure over the trunk of the portio dura of the right side: complains much of pain in the

right ear, from which there is now a free discharge of pus." With these symptoms of local affection on the right side, there were pains and numbness of the left arm and leg, and headache, but no distinct paralysis on the left side.

After this patient had been a week in the hospital she began to complain of pains in the right arm and leg, similar to those she had been suffering on the left side, and on the 13th of March all her large joints became affected with rheumatism, they were red on the surface and swollen; she sweated freely, and had a furred tongue, and her pulse rose to 100. The attack, in short, put on the characters of mild rheumatic fever. In about twelve days the fever and articular swelling were subdued, and the otitis had also improved very much. The pulse was now 80. She had so far regained power as to be able nearly completely to close the right eye. There still remained, however, considerable distortion of the face when she laughed or spoke. The discharge from the ear ceased, but some degree of deafness remained. This patient continued in the hospital till the middle of May, in all ten weeks, without any further improvement.

In consequence of the otitis, and the apparent threatening of intracranial mischief as indicated by the disturbed sensation on the opposite side of the body, mercury was at first used in this case for a short time; subsequently the rheumatic

affection was treated with sudorifics and blisters to the joints.

There can be no doubt, I think, that this was a well-marked instance of rheumatic otitis causing palsy of the portio dura, the nerve having been so much damaged that it probably would never perfectly recover its functions.

Case XVI. Charles Pottage, aged eighteen, (vol. xxxi. A.), a compositor, works a great deal by gas light; his mother is highly rheumatic, and he has himself suffered a good deal from rheumatic pains, especially of the right arm, which have weakened it very much. For six months past he has suffered very much from dyspeptic symptoms. A week before his admission his right eye became very weak and ran a great deal. He then applied to me, and I discovered all the signs of paralysis of the right portio dura, and sent him into the hospital on the 4th of January. The right side of his face exhibited all the usual signs. There was complete palsy of all the superficial muscles except the orbicularis palpebrarum, which acted slightly in winking in harmony with its fellow of the opposite side.

As this patient's general powers were very much depressed, he was treated by bark and mineral acids, and very mild aperient medicine, and liberal diet. His improvement was marked and progressive, and he was discharged cured on the 13th,

being the 9th day of the treatment, and the 16th from the commencement of the paralytic attack.

Case XVII. For the outline of the following remarkable example of paralysis of the portio dura on *both* sides, I am indebted to my friend, Mr. Holthouse, Assistant Surgeon to the Westminster Hospital.

W. B., aged forty-two, formerly a water-cress gatherer, became a patient of the Public Dispensary, Carey-street, Lincoln's Inn, in December, 1848, for rheumatic or gouty effusion into the left knee joint. As the patient was perfectly deaf and his face was paralysed on both sides to a remarkable extent, Mr. Holthouse was led to inquire into the circumstances which preceded this extraordinary amount of facial palsy.

From a very imperfect statement which the patient furnished to Mr. Holthouse, it appears that the illness which issued in this formidable face paralysis began so long ago as thirteen years. He was then seized with a severe pain in the left foot, so bad that he could not put his foot to the ground that day. In the night he was attacked with violent pain in the forehead, and that in the foot left him; soon after this he appears to have become paralysed on the right side of the face, the eye, as he describes it, staring open, and looking large and prominent. He used to suffer from pains in the head, chiefly on the

right side and in the right ear, with more or less of noise in the head. His hearing now became affected, at first so that talking to him was exquisitely painful, but afterwards deafness came on affecting both ears. A discharge now came from the right ear, and he describes the lower lip as "dropping down," and the jaw bone on both sides appearing out of place. Probably both buccinators became at this time paralysed. When he ate or spoke he was forced to put his hand to his lower lip to hold it up.

I subjoin the description of the phenomena, which this patient presents, as given me by Mr. Holthouse.

"The features generally lack expression, and their muscles are wasted; the eyes protrude, and the eyelids are farther apart than natural, so that a large portion of the sclerotic is thus exposed to view; the conjunctivæ are suffused with tears and slightly injected; winking takes place at about the usual intervals, though less completely than natural; can approximate the lids voluntarily when told to shut his eyes, but not bring them into contact one with the other, and in endeavouring to do this the eyes are rolled strongly upwards. Can neither frown nor raise the eyebrows, nor laugh, nor whistle, nor put in action any of the muscles supplied by the portio dura, unless it be the orbicularis palpebrarum to the extent already indicated. The upper lip is so elongated that its

border is on a level with the margin of the gum of the lower jaw, while the lower lip is everted and pendant as if a weight were suspended from it; by an inspiratory effort both lips can be brought into apposition and maintained so, as long as the effort is continued. The cheeks are wasted, sunken and flaccid, and the patient has lost the greater number of his teeth. The paralytic condition of the lips and cheeks, together with the loss just referred to, renders the pronunciation of the labials and dentals impossible, consequently his articulation is very imperfect, and mastication is performed with some difficulty; he is obliged to eat slowly, and to make use of his fingers as aids to the lips in keeping in the food, as well as to remove it from between the gums and the cheeks where portions of it are liable to accumulate. In drinking he presses the rim of the glass against the cuticular surface of the lower lip just below its mucous margin, whereby the pendulous free edge is lifted up and bent inwards towards the cavity of the mouth; in consequence of the lips being constantly apart, the labial glands on the lower lip can be seen to pour out their clear fluid secretion, which stands in minute and separate drops, like fine dew covering its surface; this fluid is perfectly clear, transparent, slightly viscid, neutral to test paper, and free from all organic matters as seen through an eighth-of-an-inch object-glass. Is perfectly deaf with both ears,

the loudest sounds, even through a trumpet, being quite inaudible to him; notwithstanding this high degree of cophosis, like many deaf persons he fancies he can hear, and is a regular attendant at church; can hear, he says, the playing of the organ and the rumbling of the carriages in the street, and I have satisfied myself that he is really conscious of the passing of vehicles, but it is evident that he derives the information from the vibrations conveyed to the general surface of his body, and not to the auditory organ, in other words he feels the vibrations but does not hear them; has occasional tinnitus, which he likens to the noise produced by putting a shell to the ear, "a kind of hissing noise;" sudden snappings are likewise occasionally heard in the left ear. On examining the ears, nothing abnormal is detected on the left side; but from the right meatus there flows a fœtid, purulent, and occasionally bloody discharge. A fungus growing from the cavity of the tympanum occupies the bottom of this passage, the membrana tympani being destroyed and the ossicula absent."

Case XVIII. I am induced to add another case, as a rare and instructive example of paralysis of the portio dura, in combination with temporary paralysis of a portion of the fifth nerve, and of the third, and apparently connected with secondary syphilis.

Catherine Regan, æt. 36, admitted Feb. 15th, 1845 (vol. xiv. p. 37), married, and mother of two children. In the summer of 1843 she was under surgical treatment for syphilitic periostitis of the scalp and tibia, and for iritis of the left eye. For these complaints she took mercury to salivation. In January, 1844, some tubercles formed in the subcutaneous tissue of the left arm, right leg, and right side of the face. These all suppurated; that on the face was situated in the right cheek, just in front of the ear, and burst spontaneously, leaving a deep and indelible cicatrix. Four months after this, she observed her mouth to be drawn to the left side: she noticed it while she was eating; it was not preceded by any particular pain in the head. She also found that she could not close the right eye. Soon after this her right cheek became numb, and deprived of feeling. A fortnight prior to her admission into the hospital the right eyelid dropped, and she became unable to raise it. Previous to this she suffered occasionally from headache, but since then, the headache has been constant, of a darting character, and confined to the right temporal and parietal regions; deafness has also come on in the left ear, followed by a discharge, but she never had deafness or discharge from the right ear.

The following is the description of her condition at the time of her admission, as recorded by my clinical clerk of that year, Dr. Hensley.

The face is drawn to the left side, the right cheek hangs and is very flaccid; there is ptosis of the right upper eyelid, and she is likewise unable to contract the orbicularis palpebrarum muscle of the right side, cannot purse up her lips to whistle, the right cheek flaps as she speaks or attempts to blow; the muscles of the jaw on the right side are not paralysed; there is decided loss of sensation of the right cheek and numbness of the right temple, but the eyelids and the conjunctiva are sensitive. The right side of the tongue is numb, but she can protrude the tongue straight: has no perception of the bitterness of quinine on the anterior part of the right side of the tongue, but tastes it immediately on the left side or at the base of the organ.

She complains of a constant, dull, heavy pain in the right temporal and parietal region, also of a noise in her right ear on moving the jaw, and occasionally a severe pain about the right temporo-maxillary joint. Hearing of the right ear is perfect, deafness of the left complete.

The right eyeball squints outwards and a little upwards: cannot direct the eyes in harmony, so that objects appear indistinct and sometimes double; can see better with the right than the left, probably from the previous iritis of the latter.

This patient was treated with five-grain doses of iodide of potassium thrice a day; and, in consequence of there being great tenderness on pressure

in the region of the right temple, an incision was made, on the 22nd February, down to the bone, and through the periosteum, with immediate relief to the pain of the temple. After she had been under this treatment nine days, the sensibility of the right cheek began to return, and she was able to raise the right eyelid a little. On the 4th of March, the following report was made :—she sleeps well, can now open her right eye, has less numbness of the right cheek, which, however, still hangs ; can feel when the right side of the tongue is touched : power of motion of the right eyeball is much improved. On the 18th, she could open the right eye perfectly, but could not close it.

On the 29th she was discharged, all indications of palsy of the third and fifth nerves having disappeared, but that of the seventh remaining permanent and irremediable, this latter having been, doubtless, due to the cicatrix of the suppurated tubercle.

The face is drawn to the left side, the right cheek hangs and is very flaccid; there is ptosis of the right upper eyelid, and she is likewise unable to contract the orbicularis palpebrarum muscle of the right side, cannot purse up her lips to whistle, the right cheek flaps as she speaks or attempts to blow; the muscles of the jaw on the right side are not paralysed; there is decided loss of sensation of the right cheek and numbness of the right temple, but the eyelids and the conjunctiva are sensitive. The right side of the tongue is numb, but she can protrude the tongue straight: has no perception of the bitterness of quinine on the anterior part of the right side of the tongue, but tastes it immediately on the left side or at the base of the organ.

She complains of a constant, dull, heavy pain in the right temporal and parietal region, also of a noise in her right ear on moving the jaw, and occasionally a severe pain about the right temporo-maxillary joint. Hearing of the right ear is perfect, deafness of the left complete.

The right eyeball squints outwards and a little upwards: cannot direct the eyes in harmony, so that objects appear indistinct and sometimes double; can see better with the right than the left, probably from the previous iritis of the latter.

This patient was treated with five-grain doses of iodide of potassium thrice a day; and, in consequence of there being great tenderness on pressure

in the region of the right temple, an incision was made, on the 22nd February, down to the bone, and through the periosteum, with immediate relief to the pain of the temple. After she had been under this treatment nine days, the sensibility of the right cheek began to return, and she was able to raise the right eyelid a little. On the 4th of March, the following report was made :—she sleeps well, can now open her right eye, has less numbness of the right cheek, which, however, still hangs ; can feel when the right side of the tongue is touched : power of motion of the right eyeball is much improved. On the 18th, she could open the right eye perfectly, but could not close it.

On the 29th she was discharged, all indications of palsy of the third and fifth nerves having disappeared, but that of the seventh remaining permanent and irremediable, this latter having been, doubtless, due to the cicatrix of the suppurated tubercle.

LECTURE V.

On Softening of the Brain.

I PROPOSE to call your attention to two cases of brain-disease that have recently terminated fatally in the hospital, which fatal termination gives us an opportunity of ascertaining positively what were the diseased conditions before death. A fatal termination to cases of this kind, although casting opprobrium on our art, yet affords us this consolation, that the post-mortem inspection clears away that amount of uncertainty, which overhangs most cases of brain-disease, in consequence of the various complicating sympathies that accompany disease of the central organ of the nervous system. In many cases of cerebral affection, while it is sufficiently easy to determine the nature of the disease, it is very difficult to decide upon its locality. It is only, therefore, by a careful observation of cases during life, and an unprejudiced comparison of the post-mortem changes with the symptoms, that we can arrive at accurate conclusions respecting the precise value of certain symptoms, or the exact interpretation of them. A clot in one hemisphere of the brain, encroaching more or less upon the corpus striatum, will pro-

duce symptoms exactly the same as those of a similar clot deep in the substance of the corresponding cerebellar hemisphere; and it is only by the observation and careful collation of numerous cases, made complete by their post-mortem examinations, that we can expect to arrive at such conclusions as may hereafter enable us to distinguish a paralysing clot situated in the cerebrum from a similar one situated in the cerebellum.

It is curious that in heart-disease the main difficulty is not as to the situation of the lesion, but as to its precise nature. We can easily enough tell whether this or that valve is diseased, or this or that ventricle dilated; but we cannot always be so exact as regards the precise nature of the lesion. On the whole, however, we have attained in heart-cases a certainty of diagnosis far exceeding that in brain-diseases. This we owe mainly to the much greater knowledge which we can obtain respecting the living organ from auscultation, but also to post-mortem investigations. In a large majority of heart-cases, post-mortem examinations are *corroborative;* but in brain-cases, they often give us the only clear information we possess, certainly as regards the site of the lesion; but sometimes they entirely contradict our anticipations.

Case XIX. The first case is that of William Ware (vol. xxvi.), a ploughman, from Kent, who has been in the hospital some time. He unfortu-

nately caught erysipelas, and died of it, and we had thus an opportunity of ascertaining the precise cause of his symptoms.

This man was thirty years of age, and of temperate habits. About fifteen weeks before his admission, he was suddenly seized with paralysis of the right side of his body and face. It is not certain that he lost his consciousness at first, as he woke out of his sleep in a state of paralysis. After this, according to his own account, he lost his consciousness, and continued in that state for three weeks; but it is improbable that this unconscious condition was complete coma, for coma of three weeks' duration is very unusual. He fell, perhaps, into a lethargic state, unable, as he says, to speak distinctly, or to comprehend perfectly what was said to him, or to feed himself, and deprived totally of the power of the right side.

It is always important to see what state the patient was in before the attack: in most cases you will find something wrong. Our patient had complained of a numbness of the extremities of the affected side three or four days before the attack, no doubt dependent on some morbid change going on in the brain. After the recovery of his consciousness, he continued for three weeks in a state of complete hemiplegia: he used to gape very much, and frequently cried in a childish manner, as persons thus afflicted are very apt to do. At the end of the three weeks, he began to recover

the palsy, the leg first regaining power, then the arm; but as there had been no improvement for some time, he was sent up to town.

At the time of his admission into the hospital, our patient exhibited the following symptoms:— There was hemiplegic paralysis of the right side, no longer, however, complete; for he could use the leg very well, and the arm slightly. He could elevate the arm to a right angle with the trunk, and bend the elbow-joint and flex the fingers so as to grasp feebly. In protruding the tongue it deviated, as is usual, to the paralysed side; but he could move it from side to side. The face was still slightly distorted, the cheek hanging on the right side: none of this distortion, however, was due to paralysis of the facial nerve; for all the movements of the superficial muscles were perfect: he could wink, and frown, and whistle; and when he laughed, the distortion of his face was not nearly so great as you find it when the portio dura nerve is affected. The facial palsy was due to paralysis of the buccal nerve of the fifth pair affecting the buccinator muscle. The sensibility of the affected side was slightly impaired. The muscles of the paralysed limbs were, as usual, somewhat wasted: they were, however, evidently recovering their state of tone gradually, and they were free from rigidity. Galvanism had been used several times, more experimentally than curatively, and the man was encouraged to take

as much exercise as he could without fatigue. With the notion of reducing any inflammatory process which might be going on at the seat of cerebral lesion, we gave him mercury; and while he was in a state of salivation he was exposed to the contagion of erysipelas, and was seized with that disease in a very severe form, and died.

The symptoms justify a diagnosis with regard to locality. As the paralysis was very complete, and as the motor power only was affected, it seemed in the highest degree probable that the lesion of the brain was situated in some part which exercises an important influence on voluntary motion. Now there are the best reasons for believing that no part is so intimately connected with this function as the corpus striatum. In a case, then, like the present, in which the paralysis of motion had been complete, without any considerable injury to sensation, the lesion would most probably be seated in the corpus striatum, or in that part of the hemisphere which is most intimately connected with it; and of the two corpora striata we would fix on that of the left side, since the paralysis is shown on the opposite side to that in which the cerebral disease exists; and here the paralysis was on the right side.

Taking, then, this *locality* as granted, what was the nature of the disease? It might either be occasioned by a clot of blood from a sudden rupture of a bloodvessel, *i.e.* apoplexy, or a more slow

disease, which, however, was of such a nature, as to be capable of producing sudden paralysis. The suddenness of the attack, and the succeeding loss of consciousness, favour the former supposition. But it is wrong to suppose that apoplexy is the only cause which can produce this sudden paralysis (by apoplexy I mean the rupture of a bloodvessel, and the consequent escape of blood into the brain)—there may be other causes. A portion of the brain may be undergoing a gradual process of softening for some time, and yet the continuity of its fibres may be preserved, and their constitution may not be so much altered, as to prevent them from carrying on their function as conductors, in an impaired way perhaps, but not sufficiently so as to call attention to it. Suddenly, probably from some temporary excitement, the fibres give way, and the power of voluntary motion is lost, as suddenly as the galvanic current ceases on breaking the circuit. This is one cause of paralysis in cases where there is no apoplexy.

It is now clearly established that such a lesion may often exist alone. It is, likewise, very frequently —I incline, indeed, to believe almost always—the precursor of apoplexy; and, therefore, we frequently find in these patches of white softening one or more clots of blood of various sizes. The artery or arteries leading to the part are diseased; that portion of the brain fails in its nutrition;

it passes into the state of white softening; and the minute vessels, losing the support which they must receive from the firm brain-texture, and being themselves often more or less diseased, give way, and allow the blood to escape into the tissue of the brain.*

There is another way in which paralysis may take place—viz., by an inflammatory state of the brain-substance, which produces softening, although of a different kind to that which I have just described, in which there may or may not be rupture of fibres; but there may be deposit as the result of the inflammation, which, by its pressure, may interfere with the conducting power of the fibre.

Now I have mentioned the conditions of *white* and *red* softening, or as they might be better called, *atrophic* and *inflammatory*. These are apt to come on under opposite circumstances. *White* softening may be caused by anything that diminishes or cuts off the supply of blood to the head. In some cases, in which the carotid artery has been tied for aneurysm, and the supply of blood to the brain on that side thus stopped, the patient may go on very well for a short time, and there may be no impairment of function; but during that time the process of softening has been gradually proceeding on the side of the brain which

* See further on this subject Lects. x., xi., xii.

is supplied by that carotid; and in a day or two the softened fibres cease to maintain their continuity, give way, and paralysis ensues.

Some years ago I attended, along with my friend Mr. Street, of Norwood, a very remarkable case of this sort. The supply of blood had been cut off by a dissecting aneurysm, which had plugged up the common carotid artery on the right side, and paralysis on the left side of the body took place. We were much puzzled to account for the paralysis till after death, when the post-mortem examination cleared it up. The account of this case was published in the twenty-seventh volume of the *Medico-Chirurgical Transactions*.

There is abundant evidence to show that under ordinary circumstances white softening is *atrophic* —*i. e.* dependent on imperfect nourishment of the brain, and non-inflammatory. But it may exist on the confines of genuine inflammation, the nutrient fluid being diverted from it to the inflamed portion, or it may exist around an effusion of liquid in the ventricles, that effusion being possibly inflammatory in its nature, or around an indurated portion of brain, or a tumour.

The second kind, the *red* softening, is inflammatory. I show you here a very good representation of it, which was made from a case of mine some time since. The portion of the brain affected was of considerable extent; there was

paralysis, of course, on the opposite side, a stupid, comatose condition, but not complete coma, and loss of sensibility, which continued till the patient died. But the paralysis came on gradually, and not till after these comatose symptoms had existed some time.

Now of these two conditions I think we may very justly attribute the symptoms in our patient Ware's case to a white softening, followed by a rupture of bloodvessels, and an effusion of blood.

The numbness, which he described as having occurred before the paralysis, indicated that morbid changes were slowly going on, and after a time rupture of fibres, and giving way of the bloodvessels, took place, when followed the paralysis and the three weeks' stupor. The former being very complete, denoted a solution of continuity in some part of the centre of volition; the sudden stupor implied that pressure existed within the cranium, but only to a moderate extent, for a large effusion, capable of producing such a complete paralysis, would undoubtedly have caused profound coma.

This, I say, seems the *most probable* train of morbid processes, at the commencement of the case; but we cannot speak on this subject with great certainty, for we cannot fully depend on the accuracy of the patient's history of himself. It tallies, however, very well with what we observed of his subsequent history, and of the post-mortem examination.

A train of phenomena, however, very similar, but differing as regards the *sudden* supervention of the paralytic state, might have occurred, when the primary lesion was of the inflammatory kind.

Upon examination of the brain after death, it was found that a considerable portion of the corpus striatum of the left side was completely destroyed and excavated, and that the cavity was filled with a creamy fluid, having somewhat the appearance of pus: the anterior and inner part of the corpus striatum was healthy, but the whole of the posterior and outer part was thus disorganised: a few bands of fibres, easily broken down, passed from one side of the cavity to the other. The creamy matter contained in the cavity was found on microscopical examination to contain great numbers of large cells, containing oily matter in large globules, and also in a state of extremely minute subdivision. These curious organic globules might suggest the idea, that some active process had been going on during life. What their precise signification is, I do not pretend to determine; but I think I may affirm that they are characteristic of a state of white softening of some duration, as I have found them in other cases, in which no doubt could exist of the atrophic nature of the lesion.

It seems almost certain, then, that this excavation of a portion of the corpus striatum must have been due to a white softening, followed by an

effusion of blood, and which was the immediate cause of the breaking down of the fibres and of the pressure which gave rise to the paralysis and coma. No traces of blood remained in the diseased part, as no doubt there had been ample time for the disintegration of its particles, and for its absorption.

At first the paralysis was very complete, but afterwards the patient recovered a certain amount of power, especially in the lower extremity. If the paralysis had been accompanied with rigidity, I should have been led to the conclusion that the cerebral lesion was of an irritating nature. This rigid state of the paralysed limb (*when it comes on at the same time as, or very soon after, the paralysis*) is generally seen when some superficial part is affected, as the meninges, or the surface of the brain, or when there is a growth from the skull, or a tumour in the hemispheres, in some cases of inflammatory softening, or in some conditions keeping up a constant irritation; but when there is simple rupture of the fibres of a deep-seated part of the brain, as the corpus striatum, with or without pressure, there is no irritation, and the paralysed muscles are quite lax.

There was no appearance whatever of rigidity in the muscles of the paralysed limbs. Now this is just the sort of paralysis (accompanied as it was by comatose symptoms) which would arise from a solution of continuity of fibres in the first instance,

and from compression. A portion of the corpus striatum is destroyed, and the healthy part is compressed by the effused blood. On the absorption of the latter a certain amount of power had returned in the limbs; and it depended on the possibility of repairing the broken-down portion, whether a complete restoration would take place. That parts so much diseased would have ever been completely repaired seems extremely unlikely, for two reasons—first, because the arteries of the part did not seem in a perfectly healthy condition; and, secondly, because of the well-known tardiness of all reparative processes in the brain, where actual solution of continuity has taken place.

You remember that on several occasions we passed the galvanic current through the paralytic and the sound limbs in this case. This was done, as I explained to you at the time, for the purpose of ascertaining whether any irritant disease existed within the cranium at the seat of the paralysing lesion.

If, on passing the galvanic current, you produce less contraction in the paralysed than in the sound side, then you may judge the cause to be of a *depressing* kind: if, on the other hand, the contractions in the diseased limb are the strongest, then you may conclude that the condition of the centre which causes the paralysis is *irritative*. But then you must bear in mind that irritation is not always inflammatory.

The most important points in this case may be thus summed up:

First, through some diseased state of the assimilative process, the arteries of the brain become diseased, and an insidious gradual process of softening takes places; rupture of one or more bloodvessels follows upon this, with solution of continuity of fibres, and compression of the neighbouring healthy brain-structure; after this we have absorption of the clot, and more or less attempt at reparation: it may be that some inflammation may take place around the clots, which may retard the process of recovery.

If you were called upon to treat a case of this kind from the beginning, what course would you adopt? If you can clearly make out that the lesion is not inflammatory, but, on the contrary, due to defective nutrition, the less you interfere the better. Keep the patient in the horizontal position; let the head be kept cool; unload the bowels in such a way as will involve the least effort on the part of the patient: a large turpentine and castor-oil clyster is generally more expeditious than purgatives given by the mouth; but it may be often advisable to give such a purgative as croton-oil, which is easily introduced into the system and operates freely and quickly. Bleeding is not advisable in such cases as this, as it tends to increase the atrophic condition of the brain, and

would, under such circumstances, favour rather than repress hæmorrhage.

When this patient came under treatment in the hospital, we did not at first administer any medicinal agent: he was kept quiet and nourished moderately. After one or two trials with the galvanism we observed, on one occasion, a little more excitability in the palsied limbs than in the sound under the influence of the inverse current. Finding that he had made no progress for some days, I was led, from this effect of the galvanism, to fear that some inflammation might have set up around the lesion; and, accordingly, I was induced to give him drachm doses of the solution of the bichloride of mercury—that is, as you know, the sixteenth of a grain of the salt, three times a day. No good effect followed this treatment: on the contrary, I fear the salivation, which took place sooner than might have been expected, may have made him more susceptible of the poison of erysipelas.

After all, I must acknowledge that in this part of the treatment somewhat of the *nimia medici diligentia* was exhibited. It is a lesson hard to learn, and more difficult to act upon, that nature can do more than the physician; but it is a lesson which each succeeding year of increasing experience will impress upon you, and in no cases more than in those of chronic affections of the brain.

I had intended to have brought under your

notice to-day another case of the same nature as Ware's, in which apoplectic effusions took place on both sides; but, as I fear I could not do so without making this lecture too long, I shall make this case the subject of my next lecture.

LECTURE VI.

On a Case of White or Atrophic Softening of the Brain, causing double Apoplexy.

I PROPOSE to-day to offer you some remarks on the second case which was to have formed part of the subject of my last lecture. It is one of the same nature as that on which I last commented, and it serves to illustrate the mode in which white softening of the brain is apt to take place, and the way in which that disease favours the production of apoplexy. We are fortunate in having a very full account of the case, taken with the most praiseworthy precision, by my clinical clerk, Mr. Vaux.

CASE XX. The subject of this case was named George Regan, aged 59 (vol. xxiv.); and was admitted into Sutherland ward, 20th of January, 1849. He was a glasscutter by trade, and had lived well all his life; he called himself temperate, but admitted that he had been in the habit of drinking a good deal of beer and spirits.

This man seems to have suffered long from what he called rheumatism, but which, from his habits and his age, I should think was rather of the nature of gout; he had, however, no regular

fit of gout, but complained of pains in various situations, and for these he was for some time an out-patient at the hospital.

About two years before his admission, he was seized suddenly while at work, at nine o'clock in the morning, with a feeling of stupor: as he happened to be working at home at the time, he laid down on his bed, hoping to sleep it off by dinner-time, but when he attempted to get up, he fell, and found that he had lost the use of his right side; he then became comatose, and so continued for some days. Whilst in this state of insensibility he was taken to a neighbouring hospital, where he remained for two or three months, and at the end of that time in some degree recovered from his attack, but not so as to enable him to work as he had done before his illness.

During last October he had a *second* attack, of the same kind as the first, but less severe; the limbs were paralysed, as before, on the right side; he remained a few days in bed, and recovered in some degree the use of the arm and leg, but he has never been able to work since.

On Christmas-day last, at nine o'clock in the morning, before he had risen, he had a *third* attack; on this occasion he was delirious for a short time, and afterwards became insensible, but his consciousness returned in the evening.

This third attack brought on an increase of the paralysis on the right side, so that on this occa-

sion not only was the motor power affected, but the sensibility likewise. This latter function was so far affected, that he could not pick up small objects, and he would frequently let fall things which he meant to retain in his hand, and he staggered when he attempted to walk. It was more than three weeks after this attack when he was admitted, and the paralytic state had not improved; on the contrary, he thought it worse. His condition on admission was as follows: There was imperfect palsy of the right side, as shown by slight ptosis of the right upper eyelid, and a slight hanging of the cheek, the features being a little drawn to the *left* side. He would protrude his tongue straight, but his articulation seemed somewhat difficult. In walking, he slightly dragged the right leg; the grasp of the right hand was moderately firm, but not *so* firm as that of the left; the muscles of the right leg and arm were flaccid, and less nourished than on the left side. The impulse of the heart was very strong, and a mitral systolic bellows sound could be distinctly heard.

For some days after his admission he suffered very much from constipated bowels, which it required the strongest purgatives to overcome. He then began to complain of heaviness of the head, a disposition in his thoughts to wander, and a difficulty in collecting them. These symptoms appeared to me to portend the approach of another

improvement, as is generally the case with the epileptic palsy. As this attack occurred at a time when Christmas festivities are more or less prevalent with all ranks and classes, it is very likely that the immediate exciting cause of the attack was due to over-indulgence of some kind.

The fourth attack had in its premonitory signs all the characters of a threatening epileptic paroxysm; and knowing, as we did, the existence of renal disease, we were prepared for it. The epileptic coma, however, soon passed into the profounder coma of a compressed brain; and as a new hemiplegic paralysis of a very complete kind showed itself on the left side instead of the right, it was easy to infer that a new and extensive apoplectic effusion must have taken place on the right side of the brain.

Assuming that the first three attacks were epileptic, followed by an apoplectic effusion, and that this effusion was due to a weakened condition of the arterial coats as the result of disease, it was quite consonant with experience to attribute the fourth attack to a similar cause, and to infer that arteries similarly diseased had given way on the right side of the brain. Most cases of rupture of vessels in the brain, at the age of this patient, take place from disease of the arteries; and it is very common, as was first pointed out by Bizot, for the arteries of the brain to be affected in a symmetrical manner—*i. e.* corresponding arteries

of opposite sides will be similarly diseased, and to nearly the same extent. In this way disease had been, for some time, making progress in this man's brain symmetrically; there was first palsy of the right side, and then a similar condition of the left; and this simple fact of symmetry pointed to the arterial system as the seat of disease, and therefore to its usual result, apoplexy.

On opening the body we found there was a double apoplexy corresponding to the double paralysis—one of long standing, and one recent—the former on the left side, the latter on the right. The original hæmorrhage affected the corpus striatum and optic thalamus on the left side; and it was evident that the hæmorrhage on this side did not take place at once, but on two occasions at least. The substance of the corpus striatum had evidently been the seat of the older effusion. It exhibited on section several bloody clots, and that peculiar yellow discoloration which always succeeds to a hæmorrhage. The more recent effusion was a clot which had formed quite on the surface of the optic thalamus, extending likewise to the corpus striatum, and lodged in a depression on the surface of those bodies. We had thus an explanation of the cause of the original paralysis of the left side; and its imperfect nature was plainly due to the fact, that there was but little destruction of the corpus striatum, and that the compression of the later clot affected chiefly the

attack, such as he had on three former occasions. His remarkably sallow complexion and general leucophlegmatic appearance led me to suspect the existence of renal disease, resulting from that particular form of kidney—small and atrophied—which so frequently accompanies a gouty condition. This opinion was confirmed by the characters of the urine, which was pale, of low specific gravity, and slightly but distinctly albuminous. With this view, and imagining that the uneliminated urea might be contaminating the blood and affecting the brain, I ordered him to be freely blistered at the back of the neck. This seems to have somewhat relieved him, for the next day he was reported to feel rather easier as to his head, and to have more power over his thoughts. At nine o'clock, however, on the following morning, the house-physician, Dr. Armitage, was called to him, in consequence of his having been suddenly seized with a fit; he found him completely paralysed on the *left* side, both as to sensibility and voluntary motion; his left eye squinted, and was twisted downwards and inwards; at every expiration, his left cheek puffed out, from want of power of the buccinator; the right leg and arm moved when pricked, but the left remained motionless; his breathing became louder and more stertorous, his coma deeper, and at last he died.

There was in the attacks to which this patient was subject a curious combination of the epileptic

and apoplectic, the one following upon the other. At the foundation of them, no doubt, was the diseased state of kidney. The first attack of sudden stupor was probably a slight epileptic seizure, the effect of which was a disturbance in the circulation in the brain, and the giving way of some small vessels—a slight apoplexy, with compression and rupture of fibres, and consequent paralysis.

Whilst the one or two small clots which had been effused on this occasion were undergoing absorption, and some attempt at reparation was taking place, he had another seizure of the epileptic kind,—a further disturbance of the circulation and nutrition of the brain on the same side, probably in the corpus striatum, or among the fibres which pass from it to the hemisphere. But as the paralytic state appears to have been only slightly increased, and as the coma was of short duration, it is not likely that any effusion of blood took place on this occasion.

The epileptic character of the third attack was manifested in the delirium with which it was ushered in, and which ended in coma. There was a decidedly increased paralysis after this attack; but since quite as much paralysis is apt to follow the simple epileptic seizure, it by no means follows that any effusion of blood took place, although it is probable that such must have been the case, from the fact that the palsy showed no signs of

improvement, as is generally the case with the epileptic palsy. As this attack occurred at a time when Christmas festivities are more or less prevalent with all ranks and classes, it is very likely that the immediate exciting cause of the attack was due to over-indulgence of some kind.

The fourth attack had in its premonitory signs all the characters of a threatening epileptic paroxysm; and knowing, as we did, the existence of renal disease, we were prepared for it. The epileptic coma, however, soon passed into the profounder coma of a compressed brain; and as a new hemiplegic paralysis of a very complete kind showed itself on the left side instead of the right, it was easy to infer that a new and extensive apoplectic effusion must have taken place on the right side of the brain.

Assuming that the first three attacks were epileptic, followed by an apoplectic effusion, and that this effusion was due to a weakened condition of the arterial coats as the result of disease, it was quite consonant with experience to attribute the fourth attack to a similar cause, and to infer that arteries similarly diseased had given way on the right side of the brain. Most cases of rupture of vessels in the brain, at the age of this patient, take place from disease of the arteries; and it is very common, as was first pointed out by Bizot, for the arteries of the brain to be affected in a symmetrical manner—*i. e.* corresponding arteries

opposite sides will be similarly diseased, and to
arly the same extent. In this way disease had
en, for some time, making progress in this
n's brain symmetrically; there was first palsy
the right side, and then a similar condition of
e left; and this simple fact of symmetry pointed
the arterial system as the seat of disease, and
erefore to its usual result, apoplexy.

On opening the body we found there was a
uble apoplexy corresponding to the double para-
is—one of long standing, and one recent—the
mer on the left side, the latter on the right.
e original hæmorrhage affected the corpus stri-
m and optic thalamus on the left side; and it
s evident that the hæmorrhage on this side did
t take place at once, but on two occasions at
st. The substance of the corpus striatum had
dently been the seat of the older effusion. It
nibited on section several bloody clots, and that
culiar yellow discoloration which always suc-
eds to a hæmorrhage. The more recent effusion
s a clot which had formed quite on the surface
the optic thalamus, extending likewise to the
rpus striatum, and lodged in a depression on
e surface of those bodies. We had thus an ex-
nation of the cause of the original paralysis of
e left side; and its imperfect nature was plainly
e to the fact, that there was but little destruc-
n of the corpus striatum, and that the com-
ession of the later clot affected chiefly the

optic thalamus, and but slightly the corpus striatum.

The apoplectic effusion on the right side was much more extensive; it involved parts corresponding to those affected in the first seizures, but to a much greater extent; and the brain substance was more completely torn up and destroyed. The clot was very large; it filled the right lateral ventricle, and even broke through its roof, and tore up the white substance of the cerebral hemisphere: it likewise broke down the corpus striatum, optic thalamus, and the septum lucidum, all which parts were completely swept away. This remarkable destruction was no doubt owing to a previous diseased state of the brain. I mentioned to you in my last lecture that it is very common for apoplectic effusions to be preceded by white softening; and when the softening has got to a certain extent, the brain substance no longer affords an adequate support to the vessels, which, themselves more or less enfeebled by disease, give way, not in one point only, but in many, and the abundant effusion ploughs up the softened matter, quite destroys it, and takes its place: all this was the case in the present instance. The arteries of the brain were very generally diseased on both sides, and exhibited that symmetry to which I have alluded.

We found likewise, as had been anticipated, a diseased state of the kidneys: they were very much contracted, the cortical substance wasted,

fissured, and granulated on its surface, the tubular substance healthy, and the capsule thickened. This condition of kidney, formerly described as the third stage of Bright's disease, is, in reality, a chronic degeneration or wasting of the kidneys, due to a deranged and damaged nutrition, for which I should be glad to find some other name than chronic nephritis: I have called it *gouty kidney*;* and in this and many other cases this name is very appropriate. But it occurs in cases where there is no evidence of gout. The result of the disease is to render the kidneys imperfect emunctories for the elimination of the urea, and other elements of the urine, which accumulate in the blood, and give rise to various morbid changes throughout the body, and are especially mischievous to the functions of the brain.

The heart and the arterial system were likewise extensively diseased. In the coats of the arteries were very numerous deposits of atheromatous matter. The heart was much dilated and hypertrophied, especially as regards the left ventricle. The fibrous basis of the valves was extensively thickened, apparently by some deposit, which rendered it opaque, and impaired its flexibility; and as is generally the case in this particular form of disease, *all* the valves were altered in this way;

* See on this subject the Lecture on Gouty Kidney in my volume of Clinical Lectures "On certain Diseases of the Urinary Organs, and on Dropsies."

those of the right side, however, being much less diseased than those of the left. The semilunar valves of the arteries, especially of the aorta, had the fibrous festoons at their basis much thickened, the curtain of each valve being very little affected, and its functions, therefore, not impaired; and the cordæ tendineæ of the mitral valve were much thickened, and somewhat shortened, and the curtains of the valve also thickened. Similar changes had taken place in the tricuspid valve and its tendinous cords, but to a much less extent.

Taking, then, a general retrospect of this case, we find there is quite enough to account for all we have seen. The sequence of the events may be thus described:—First, the man gets into a general gouty condition, and the elimination of this morbid material gives rise to an irritation of the kidney, which at length assumes the form of gouty kidney, or, if you will, chronic nephritis; and the kidney, thus damaged and incapacitated for the perfect discharge of its function, is the great promoter of all the subsequent evils: the blood becomes still further contaminated, additional deposits to those which doubtless had already formed take place in the tissue of the heart's valves, in the large systemic arteries, and in those of the brain; the diseased arteries of the brain become insufficient channels of supply; white softening is the consequence, and many of the

unsupported and unhealthy capillaries at length give way; and thus all the circumstances, from first to last, fall in regular order as cause and effect.

The deposits in the arteries produce a twofold influence upon the circulation—by roughening the inner surface of the arterial channels they create a certain amount of direct obstacle to the flow of blood from the ventricle; and by diminishing, or nearly destroying, the elasticity of the arterial walls, they impair one of the most important forces by which the circulation is carried on in the arterial system. Thus the arteries, from being elastic yielding channels, with perfectly smooth inner surfaces, are changed into resisting inert tubes, with rough interiors. It is plain, then, that, under these circumstances, the heart has to encounter great obstacles, and to do a great deal more work than when the arteries are in their normal state; hence the dilatation of the cavities of this organ, caused by the obstacle to the free flow of the blood; and the hypertrophy, by the greater exercise and effort of its muscular fibres. The increase of force in the heart's action, however, is merely remedial, to meet the increase of obstacle, and is one of those beautiful instances of self-adaptation to change of circumstances which the animal organism, especially the muscular system, so abundantly exhibits.

As these deposits go on, they impair the structure of the arteries of the brain; the degenerated

walls of these vessels become less strong, and less able to support their contents. And, I would particularly impress upon you, that there is no excessive determination of blood to the brain, in these cases, but the reverse; for the blood that goes to the head has, in the erect posture, to be pumped up against the force of gravity; and, therefore, any obstacle in the course of the arteries would tell more in this than in any other direction. It is a common notion, too easily accepted by many excellent practical writers, that hypertrophy of the heart gives rise to apoplexy, by sending the blood with an undue impulse to the head; but for the correction of this error we need only remember that the additional force is merely such as is necessary for the exigencies of the circulation, and such as shall preserve the force of the blood's current as near as possible to the normal point, in spite of the existing obstruction. Indeed, the actual force with which the blood circulates in the morbid arteries is, most probably, less than in health. The apoplexy is, in fact, due to the diseased state of the arteries, which renders their walls an inadequate support to their contents, and to the diseased state of brain, which imperfectly supports the arteries.

A vast number of the cases of apoplexy which occur about the period of life of our patient, or after the age of 50, are of this kind—a fact that has an obvious and an important bearing upon the question of treatment.

There is a practice, unfortunately too common, but which, I think, is every day becoming less so—namely, that of following an attack of apoplexy, very much as a matter of course, by depletive measures. However, it may be, that some justification may be found for such a mode of treatment in the cases of strong, young, hale, and plethoric subjects, I presume no one will say that it is very well adapted to patients who have passed the meridian of life, whose blood and tissues are more or less contaminated by morbid matters, and with whom a diseased state of the arteries of the brain has already greatly weakened the nutrition of that organ. The case, indeed, which I have just detailed to you, is one of many, which proclaim loudly, that a depletory system ought not to be pursued indiscriminately, or even generally, in apoplectic cases.

With reference to this question of depletion in apoplexy, let me refer you to an interesting and very useful work by Mr. Copeman, in which he has collected, from a great variety of sources, a large number of cases which presented the symptoms of apoplexy. Of 155 cases in which the treatment was specified, 129 were bled, and only 26 were not. Of the 129 persons who were bled, 51 recovered, and 78 died—the recoveries being about 1 in $2\frac{1}{2}$, the deaths 1 in $1\frac{2}{3}$. Of the 26 who were not bled, 18 survived, and 8 died, the proportion of recoveries being 1 in $1\frac{1}{2}$, and of deaths 1 in $3\frac{1}{4}$. Eighty-five of the cases were

bled generally and copiously, and of these only 28 recovered, and 57 died—in other words, two in every three cases terminated fatally. I am quite aware that the small number of cases not bled casts some doubt on the validity of the conclusion to be drawn from the comparison of the results of the treatment. But the fact that considerably more than half of those treated by bleeding died (and we owe much to the industry of Mr. Copeman for bringing it out), is a highly significant one, and should arrest attention.*

I hope I have now said enough to convince you that the treatment of patients with apoplectic symptoms must not be regarded as a matter of routine, but as a question of grave import, and which demands the most anxious consideration of the practitioner. Let me add, that it sometimes requires the exercise of no small courage and self-possession to resist adopting that practice; for the popular feeling, led by a formerly too prevalent medical practice, is entirely in favour of it, and would readily condemn a practitioner as guilty of the death of his patient who suffered him to die unbled. It is a far more dashing and courageous thing to open a vein on the spot, and in the presence of a number of anxious friends, than to adopt less showy, and apparently less active, measures.

But, indeed, you need not be inactive, even if

* A Collection of Cases of Apoplexy, by E. Copeman, 1845.

you decide against adopting the plan of bleeding. Having placed your patient in an easy position, in which no excitement of muscular action is likely to take place (for you must bear in mind that reflex actions may often be readily excited in these apoplectic cases), you should immediately direct your attention to the state of the stomach and intestinal canal. Sometimes in these cases the stomach is overloaded, or the bowels are confined, and the administration of a quickly-acting emetic, or even of some purgative medicine will often provoke a moderate sickness, which unloads the former. Nor can there be any objection to adopting measures to clear out the bowels, either by an active purgative administered by the mouth, such as calomel or croton oil, or by a stimulating and purgative enema, or both.

If, upon full inquiry into all the particulars of the case, you find that your patient is of plethoric habit, with too much blood in his body, and with a sufficiently strong heart, you may bleed him with reasonable chance of benefit; but if he has been of intemperate habits, is labouring under organic disease of the heart and arteries, is of gouty or rheumatic constitution, then, whatever popular or medical custom may say, my advice to you is, hesitate much, before you deplete by bleeding.

The objects which it is proposed to gain by bleeding are a diminution of the cerebral congestion, and the stoppage of the hæmorrhage into the

brain; and where it is quite plain that cerebral congestion does exist, and that that congestion causes the cerebral hæmorrhage, this is clearly a rational practice. But you must bear in mind that in a large number of the cases—probably the majority—there is in reality no cerebral congestion, and that the hæmorrhage is of a kind not likely to be stopped by taking away blood—by establishing another hæmorrhage elsewhere.

On the whole, then, I think that the results of experience denote that the majority of cases of apoplexy are best treated by purging, shaving the head and keeping it cool—perhaps blistering, that bleeding is rarely applicable, except to the young, vigorous, strong, and plethoric, and that the practitioner should aim more at keeping the heart's action quiet than at any other single indication.

LECTURE VII.

On Softening of the Brain.

GENTLEMEN,—We can illustrate to-day the influence of disease of the brain in the production of paralysis, and also the pathological changes in that organ, which are capable of cutting off the influence of the will from a large portion of the body. For this purpose I shall request your attention to the particulars of three cases.

The first case ran so short a course that I fear many of you missed the opportunity of seeing the patient during life. The facts observed in the *post-mortem* examination of this case are of especial interest, inasmuch as they afford an explanation of the early changes which take place in the brain's structure, leading ultimately to such alterations as favour rupture of the bloodvessels, and the consequent escape of blood, in greater or less quantity, into the surrounding texture, constituting that condition which is called Apoplexy, or Sanguineous Apoplexy.

CASE XXI. The patient, by name William Thurston, (P.M. Book, page 15,) was an auctioneer's porter, a muscular, well nourished man, about the age of fifty; he had always been of in-

temperate habits, and about six months before the fit, had lost his wife; under the influence of an ill-regulated grief he was led to yield too much to self-indulgence, and to console himself with the bottle. On the morning before his admission, he fell senseless on the floor, but it did not appear that he bit his tongue during the fit. In about half an hour he recovered so much as to be able to attend to his business. It seems most probable that this was an epileptic fit, which, however, wanted the very characteristic feature of biting the tongue. That symptom, however, is not always present, and, although when it occurs along with the other symptoms, it may be regarded as pathognomonic of the epileptic fit, its absence by no means proves that the attack was not of that nature. The fact, that a patient has bitten his tongue in the attack, shows in a striking point of view the profound insensibility that accompanies the fit; for, in the insensibility of the patient to pain, the tongue is not withdrawn from between the teeth, and so it gets bitten by the violent and spasmodic closure of the jaws. In the course of the day, while at his work, our patient fell down in another fit, in which he remained quite unconscious, but did not struggle. He was brought to the hospital in a state of profound coma, at four o'clock, P.M.; his eyes were shut, and the pupils were quite insensible to the stimulus of light. The fall was so sudden and heavy that he bruised his chin, and loosened four of his teeth.

He lay in a totally unconscious state, and the breathing was accompanied with a snoring noise, not of the loudest kind. The pulse and respiration were not particularly rapid at this time, nor could any evidence of a paralysed condition of either side of the body be obtained. So things went on until another fit commenced, in about an hour after his admission, which was accompanied by violent spasmodic action of the muscles of the whole body, affecting chiefly those of the lower extremities. There was no indication that the convulsions were greater on one side of the body than on the other; this point was so closely watched that if there were any difference, it must have been very slight. The convulsions were accompanied by an acceleration of the respiration, and by increased noise in the throat; in about ten minutes the patient was quiet again, but in an hour more the same train of phenomena was repeated, with still more noise accompanying inspiration, and a puffing out of the cheek at each expiration. After this fourth fit several other attacks manifested themselves at still shorter intervals, and he died in the night, in a very exhausted state.

Now we often meet with phenomena such as have been exhibited by this patient, without being able to find any *post-mortem* appearances which will explain their occurrence; hence I gave a doubtful diagnosis. Before the body was opened,

I stated that we should find one of three conditions, first, a superficial clot extending over a great part of the surface of the brain; or, secondly, a clot in the arachnoid sac, or between the dura mater and the bone, or even between the layers of the dura mater; or, thirdly, a state of brain apparently normal, which would be quite insufficient in itself to account for the symptoms during life, in which case the epileptic condition would probably be found to have been associated with disease of the kidney. We were not able to determine the condition of the kidneys during life, as we had no opportunity of examining the urine. The difficulty of the diagnosis was much increased by the fact that one side of the body did not appear to have been more affected than the other. This point was carefully attended to by those who watched the patient whilst he was suffering from the convulsions. Had the convulsions been greater on one side, one would have been led to the inference that a clot was present in the opposite side of the brain; but in the absence of such evidence I was quite prepared to find that the convulsions were due to the change in the state of the blood which is induced by kidney disease. The symptoms, however, only extended over a period of twelve hours, which is a much shorter time than the epileptic attacks depending upon kidney disease generally take to run their course. Such cases usually last much longer

(unless the fits are very violent and at short intervals) than those in which cerebral hæmorrhage has taken place; the quantity of blood effused, which is often very considerable, tending, in the latter cases, in some measure to expedite the fatal result.

Let me, however, describe the *post-mortem* appearances in detail.

The dura mater was remarkably adherent to the calvaria, over the situation of the Pacchionian bodies, a condition, which we very frequently meet with in persons, who are much addicted to drink, or are prone to violent fits of passion, and in these cases the Pacchionian bodies themselves are often observed to be unusually large.

Upon cutting through the dura mater a large clot was found in the cavity of the arachnoid, about the situation of the squamous portion of the left temporal bone. In the posterior part of the head a much larger quantity of blood was found effused, chiefly between the hemispheres, and on the left side of the falx. The quantity could not have been less than two ounces. The pia mater over the whole surface of the brain was found to be highly injected with blood.

Now comes the most interesting by far of all the appearances which we noticed. On the superior surface of the right hemisphere, one or two circumscribed patches of grey matter were observed to be of a much darker colour than the surrounding parts. On closely examining these,

it was found, that the colour was due to the presence of a number of minute red points, which under the microscope were found to consist of little extravasations of blood into the grey matter of the brain. Upon submitting portions of these dark patches to microscopical examination, the capillaries were found to have undergone fatty degeneration, the deposits of fat (consisting of aggregations of very minute oil globules) being in the walls, and arranged in alternate patches on the sides of the vessels, apparently in the situation of development cells. Of this appearance, I show you an excellent sketch by my late clinical clerk, Mr. Curme. The white matter of the brain appeared to be healthy and of a natural consistence. The ventricles were free from effusion, as was also the base of the brain.

The depositions of fatty matter in the minute vessels may interfere mechanically with the passage of the blood through them, and they would certainly interfere with the development of the force of vital contractility, which, as you know, is present in healthy vessels, and upon which the strength of these vessels mainly depends.

No more important observation has been made of late years in minute anatomy than that which showed that the minute bloodvessels are apt to become the seat of an atrophic process, in which the normal tissue (probably the muscular) of the capillary walls is replaced in great part by fat.

It had long been noticed that the larger blood-vessels were more or less diseased in cases in which hæmorrhage had occurred on or into the brain; but it was evident that the hæmorrhage did not depend on the rupture of a large vessel, it had all the characters of a bleeding which came from several minute points, and had merged into one great clot. The sources of these small hæmorrhages are well illustrated in the condition which I have described in the right hemisphere of this patient. This fatty degeneration of the capillary vessels will hereafter take its place with other degenerations of a similar character, which modern research has brought to light; such as the fatty disease of the kidney, one of the forms of disease of that organ in which albuminous urine occurs; such likewise as fatty degeneration of the muscular fibres of the heart, in which many a case of broken heart originates.

In looking into the pathology of this condition of the minute bloodvessels, you may fairly ask, is it, indeed, the primary and essential disease? or is it not rather an effect of a depraved nutrition of the tissues to which these canals are the carriers of nutriment? Now, a little reflection would lead you to say that both these questions may be answered in the affirmative, and that the evil of which we speak may either commence in the brain-tissue (the brain-fibre or the brain-cell), which consequently cease to draw upon the bloodvessels

to their wonted extent; and the bloodvessels, therefore, deliver to them their nutrient matter in diminished quantity; their activity is, consequently, proportionably diminished, and an atrophic state ensues. Or the evil may commence in the bloodvessels; and this will probably be the favourite view. We know that the larger vessels are frequently the seat of numerous atheromatous deposits, and not only the cerebral vessels, but the aorta itself, and the radial arteries, as we can often learn by feeling the pulse in the living person. Why should not this atheromatous state extend to the minutest vessels? I do not express any strong opinion in favour of either of these views; but I would say that it seems to me that pathologists do not enough regard the tissue itself as being the starting point of morbid change; and are too apt to overlook the fact, that the power of attraction of the tissue for certain elements of the blood is not the least important agent, in determining the greater or less flow of blood to the organ or tissue.

And, indeed, whichever of these two views we adopt, who will say that in the blood itself we do not find the chief source of evil, which may tell either upon the bloodvessels or upon the elements of the tissues?

Without waiting to decide this point, we may lay it down that, whether in consequence of disease of the bloodvessels or otherwise, the nu-

trition of the brain becomes impaired, and this shows itself mainly in an altered consistence of the nervous matter. Its colour does not undergo any appreciable alteration, and if you look at a portion through the microscope you will not detect any obvious change, the vesicles remain the same and the fibres remain unchanged; but, as I have said, the consistence is diminished, instead of being firm, the tissue of the organ has become soft and pulpy, and in some cases almost diffluent and of the consistence of cream. The diseased blood-vessels lie in the midst of this pulpy mass for some time without undergoing any further change, but sooner or later, under some mental emotion, or during some increased heart's action depending either upon a strong mental emotion, or an increased mental effort of any kind, or upon derangement of the digestive organs, or upon some bodily exertion, the blood is sent with undue force or in unusual quantity into the vessels, and in consequence the vascular canals in the pulpy portion of the cerebral tissue being deprived of their usual support, give way, and blood is effused into the softened part of the brain, which it breaks up, and the more readily in consequence of its already diminished consistence.* This is the *rationale* of the development of many an attack of apoplexy,

* An epileptic fit, by retarding the venous circulation, increases the blood pressure in the arteries, and is, I have no doubt, a very common cause of cerebral hæmorrhage.

from which the patient may or may not recover, according to the extent of brain previously softened, and according to the amount of blood effused.

In the case of Thurston, the presence of the red points above described afforded evidence of the occurrence of small extravasations. Other vessels in the pia mater were doubtless in a similar condition to those of the right side, and these giving way in the manner I just now referred to, led to the extensive effusion of blood, which we met with on the surface of the brain. Had the extravasations been limited to the dark patches of grey matter, I should imagine that the attack would have been but slight and transient, and the patient might for a time, at least, have recovered. Such cases we often meet with, when the only symptom is a transient confusion of ideas, or perhaps only a sensation of faintness or giddiness; after a lapse of a longer or shorter period, this slight attack is followed by one of a more serious nature, in which loss of consciousness, or paralysis, or both may occur.

We had no evidence in this case, during life, that any paralysis existed, or that the convulsions were excited more on one side than on the other. This was sufficiently explained by the *post-mortem* appearances. The lesion was too much on the surface to cause paralysis, and the compression caused by the effusion of blood was exerted over

an extended surface, but not to any great depth. The clot, by extending between the hemispheres, compressed both, and although it was larger over the left hemisphere, the right was more diseased, and was itself the seat of small effusions in some of its convolutions.

In such cases as this, the paralysis, when it occurs, will be due to the solution of continuity of fibres connected with the corpus striatum, or optic thalamus, or to compression, direct or indirect, of those bodies; the loss of consciousness will arise from the effusion of blood, and the consequent compression of and shock to sound parts of the brain. It is, I think, not unreasonable to suppose that a simple solution of continuity of brain fibre may occur without any extravasation of blood; and there are cases of paralysis which occur under these circumstances to which I shall take another opportunity of calling your attention. But I suspect that, in every case where consciousness is affected, and where more or less of coma takes place, there will be found more or less of extravasation of blood, excepting always uncomplicated cases of renal disease and of true epilepsy, or cases in which the softening involves a very large portion of the convoluted surface, or of the white substance of the hemispheres of the brain.

I need scarcely tell you that the condition of brain to which I allude is called white softening.

This disease, when connected with a morbid state of bloodvessels, occurs most frequently at the more advanced periods of life, and is essentially an atrophic condition. After some time, the diseased brain-substance exhibits not only the diminished consistence which I have mentioned, but also the development of a peculiar series of large cells filled with fatty matter. These cells, as they occur in the more chronic cases, are generally found in the immediate vicinity of capillary vessels, and sometimes are disposed, in a very striking manner, in rows parallel to them; they may perhaps be interpreted as abortive attempts at new fibres, or as products of some change in the secondary destructive or assimilative processes.

There is one other point which I must briefly notice in the history of this case, namely, that the fatal symptoms were ushered in by an attack distinctly epileptic in its character, from which the patient perfectly recovered, nor did a fresh attack come on until some hours after. I think it important that you should especially notice these cases of epileptic paroxysms coming on in states of brain in which the supply of blood is rather deficient than superabundant.* The fact has an interesting bearing on the pathology of epilepsy in general.

* Vide Case XX. Lect. vi.

Let me now pass on to another case which affords a good illustration of the *symptoms* which accompany this lesion during life, while the case, just related, illustrates the morbid anatomy.

Case XXII. H. B., æt. 72 (vol. xxxvii., p. 64). Our patient is a full, plethoric man, the beau ideal of the popular conception of an apoplectic subject, with a short neck, and a rubicund countenance. He has seen much better days, and, indeed, for many years represented an important constituency in parliament. He used to live well in the common sense of the expression, and to enjoy the good things of this life while he could get them. But he acted too much on the principle "let us eat and drink, for to-morrow we die." In short, he has been a jolly, hearty sort of fellow, who has lived a dissipated and a careless life, and who latterly has had to undergo much privation and distress, interlarded every now and then with a bout of intemperance to drive away care. On the 15th of June, while walking in the street, he suddenly became giddy, and fell backwards to the ground. He had experienced a transient feeling of giddiness once or twice within the same week, but did not fall on either of these occasions. He became lethargic and stupid, but retained consciousness when the fit occurred, for he informed us he could distinctly recollect a medical man coming up to bleed him, and he also felt the prick

of the lancet. When taken up, he was found to have lost all voluntary power over the left side, and he was unable to speak; but sensibility was not destroyed in the affected side. He remained under treatment in his lodgings until the 21st, when he was brought into the hospital, and the following note was made of his condition at that time:—

There is no power of motion on the left side; but the sensibility of that side is unimpaired. The left cheek hangs, and the face is drawn to the right. The tongue, when protruded, moves to the left side. The muscles of the paralysed limbs are in a relaxed state, excepting the biceps muscle of the arm, which is slightly rigid, and becomes more manifestly so when you attempt to extend the forearm upon the arm. His articulation is imperfect, and he cannot swallow solid food, although fluids pass down readily.

The artery at the wrist is found to be in a thickened state from atheromatous patches. In all cases of apoplexy, especially in advanced age, you should try to ascertain the actual condition of the radial artery, as regards these deposits. If this vessel be freely studded with deposits, it is highly probable that other arteries are so too, and not the least likely, those of the brain. The heart's sounds are feeble, as if the nutrition of the muscular substance of the organ had been impaired, and, upon listening carefully, a roughness

accompanying the first sound is heard at the apex; the pulse is intermittent in character. Being very much depressed on his admission, the patient was ordered to take small and frequent doses of ammonia, and suitable nourishment.

There is a point connected with this case to which I may refer briefly. On examining the cornea of the eyes, we found a large and well-marked *arcus senilis* around each cornea. This condition has attracted some attention of late, in consequence of Mr. Canton having shown that it is a fatty degeneration of the cornea commencing at the circumference. It occurs, as we had long known, generally after fifty; sometimes, though rarely, at an earlier period. Sometimes it encroaches very largely upon the cornea. I know a gentleman of seventy-eight, in whom it forms nearly a third of that tunic. It is supposed to have a connexion with fatty degeneration of the arteries, and with fatty heart; but it by no means follows that because the first exists, the others should also; although no doubt the three often co-exist. And if you find a thickened condition of the radial arteries at the wrists, with indications of atheromatous deposit and a large arcus senilis too, it would be highly probable that the fatty degeneration of the small arteries of the brain had taken place, and this suspicion would be greatly increased if there have been an apoplectic attack.

Our patient, H. B., has improved slightly since

his admission. He is now (June 29th) much less lethargic, and has acquired some power in the paralysed limbs. Reflex action is more easily excited in the lower extremity on stimulating the sole of the foot.

What is the diagnosis in such a case as that of H. B.?

The case is not likely to be confounded with epilepsy, because there has been no absolute suspension of consciousness from the first moment of the attack. I think the phenomena of the case are best explained as follows:—

There have been taking place, very gradually, and probably over a long period of time, deposits in the larger arteries of the brain, impairing more or less the nutrition of the parts which are supplied by these vessels, and generating a soft and friable condition of the nervous matter. Ere long a decided hæmorrhage takes place which breaks down the soft cerebral matter, severs a large number of fibres, and creates shock by the suddenness of the laceration of the brain substance, and by the compression of the healthy parts of the organ, consequent upon the escape of the blood from its normal channels. The result of the combined influence of shock and compression is the development of a more or less comatose state, which is, in the main, proportioned to the quantity of blood extravasated, although also to a degree dependent on the position of the clot.

And the effect of the rupture of a large number of nerve-fibres is to disconnect the centre of volition from a greater or less portion of the body, according to the number of nerve fibres lacerated.

It is important, however, to bear in mind that an equal amount of paralysis might be induced without any laceration of brain, or rupture of fibres. The compression of the surface of the brain would produce this effect, but for this purpose the clot must be a large one. There are two points that render it improbable that, in the case under discussion, the lesion is of this nature; first, that a clot of sufficient magnitude to cause by simple compression so much paralysis, would probably give rise to a much greater amount of coma than existed; and secondly, that so much compression of the brain would be accompanied with much greater rigidity of the paralysed muscles.

Can we form any exact opinion as regards the precise position of the paralysing lesion in the brain? I have already stated why I do not believe it to be a clot on the surface. From the completeness and extent of the paralysis, it seems probable that a considerable portion of the centre of volition is in some way interfered with. Assuming the lesion to be white softening, and a clot, with ruptured and torn brain fibres, it may be situated deep in the hemisphere, just outside the corpus striatum, and perhaps encroaching upon it,

or in the corpus striatum, or the optic thalamus, or in both, or in the crus cerebri, or deep in the substance of one hemisphere of the cerebellum.

This patient remained in the hospital for nearly four months. The paralysed limbs had gained but a slight degree of power; but consciousness was completely restored; he never, however, gained strength; he became imbecile, and dirty in his habits, and extremely restless. His friends removed him into lodgings, where he soon died. An examination of the body was not allowed.

I must hasten to notice a third case, of which I have only time to take a brief survey. It affords a good example of what appears to me to be simple white softening with solution of continuity of fibres, and without clot.

Case XXIII. Comfort Winning, aged sixty-one. Lonsdale ward, admitted June 16, (vol. xxxvi., p. 76.) This woman had several confinements, and some severe ones; and she appears to have suffered from bronchitis in September, from which, however, she recovered perfectly. In February last she lost the use of the *left* side of the body suddenly, and without affection of consciousness. The paralysis, according to her statement, was not complete, and she recovered perfectly within three months. Exactly twenty-four days before her admission she had a second attack. She was sitting at breakfast, and suddenly lost the use of the

right arm and leg, with no other affection of consciousness than a sense of confusion and giddiness, and some pain in the head. This time the paralysis was very perfect; the arm and leg were completely paralysed as regards motion, sensation being slightly affected; the right side of the face was paralysed in the ordinary way. Her articulation also was much affected.

When admitted into the hospital, there was still palsy of the right arm; the leg had acquired a little power, she could move it slightly, and reflex actions were readily excited under the influence of stimulation of the sole of the foot. The palsy of the tongue and face had recovered, and the speech was less affected. Common sensation was but slightly affected; but she suffered a great deal of pain in the paralysed limbs, especially in the arm, and there was a feeling as if the gums were swollen and the tongue too large for her mouth.

The paralysed limbs are completely relaxed. There is no rigidity of the muscles whatever, and the forearm may be flexed or extended on the arm without the least resistance. The fingers are slightly flexed towards the palm, and offer a little resistance to extension. There is a distinct arcus senilis in each cornea. The pulse is natural, but feeble, and there is no evidence of disease of the heart.

This woman has now (June 29) been thirteen days in the hospital. During that time she has

manifested little or no improvement. She has suffered chiefly from a very distressing symptom of not uncommon occurrence in similar cases, namely, severe pain in the paralysed limbs—partly referred to the joints, partly to the course of the principal nerves of the limbs. These pains are most severe in the arm, and are most troublesome at night, interfering much with rest. The only advance of power is seen in the leg.

This case is in all probability one of white softening from obstructed circulation by diseased arteries. There is probably no clot, or at most a a very small one. The grounds upon which I have formed this opinion are these: First, the age of the patient, which is favourable to the morbid change of the vessels with which white softening is so often associated. Secondly, the fact that the present is a second attack, on the opposite side to the first. You know that, as was first made out by Bizot, the cerebral bloodvessels are apt to become diseased symmetrically. The former attack was probably due to a diseased state of bloodvessels; if there have been diseased vessels on the right side, it is probable such exist on the left, and this second attack might have been predicted with some degree of probability on the occasion of the first. Thirdly. The arcus senilis is well marked in each cornea.

The recovery from the first attack was in all probability due to a collateral circulation, which

was sufficient to restore the nutrition of the softened fibres and to heal the breach. This attack, however, was comparatively slight, and it is not likely that much of the brain was involved. The second attack has evidently resulted from a much more extensive lesion.

I think you will admit that it is improbable there has been any extensive effusion of blood in this case, or, indeed, any effusion at all, if you consider that a clot of sufficient size to create all this paralysis would inevitably have caused more or less coma. The suddenness of the occurrence of the palsy appears to me to be sufficiently accounted for, on the supposition that a number of fibres connected with the centre of volition became softened up to a certain point, and giving way suddenly by a sort of deliquescence, severed the communication between the centre of volition and one half of the body.

The prognosis respecting this case is not favourable as regards the recovery of power in the limbs. A second attack, involving a large extent of brain, is not likely to be recovered from, because a considerable number of bloodvessels are doubtless diseased; and it is not probable that a sufficient collateral circulation can be obtained to bring the requisite supplies, through other channels, to the diseased portion of brain. But as to the duration of life, it is difficult to assign the period, as that will depend upon general care and nourishment,

and the absence of causes which may unduly excite either the cerebral or the general circulation.

The treatment adopted in this case was simply that of supporting by an appropriate and rather generous diet. Quinine was administered for a short time, and she took also iodide of potassium in five-grain doses every night with benefit to the pains in her limbs.

[This patient remained in the hospital till the end of July, and left improved in general health and as to the power of the leg, but without any increase of power in the arm.]

LECTURE VIII.

On a Case exhibiting Cerebral Symptoms connected with Renal Disease, and terminating in Sanguineous Apoplexy; and on a Case of Delirium.

Gentlemen,—Two cases have within the last day or two terminated fatally in the wards of this hospital; these I mean to make the subject of my observations to you to-day: they are both of a very interesting nature, highly illustrative of important points in pathology, and such as will well repay careful study by those who have narrowly watched them.

Case XXIV. The first is that of Moses Jeffrey (vol. xxvi., p. 56), a heavy, lethargic patient, whom you must all remember as having been long an inmate of the Sutherland ward, where he was admitted on March 7th, 1849. His disease belongs to an interesting class of cases, which, while they exhibit a remarkable similarity of symptoms, may be referred to very different causes. Our knowledge of the real nature of these cases is deduced from the union of the study of clinical history with post-mortem examination. Indeed, without the opportunity of examining the state of the principal organs of the body after death, we should

never have arrived at any accurate knowledge of the real nature of these cases; for, did we look alone to the symptoms we should be led to refer them to only one and the same cause—as, indeed, had always been done, until modern researches, by means of that union to which I have already referred, of diligent clinical study with post-mortem investigation, taught us to interpret the symptoms, and to appreciate their real value and indications.

Our patient was one of a class of men addicted to intemperate habits; he was a compositor by trade: he had formerly drank a great deal, but for eight years he had been a teetotaller, and had strictly adhered to the pledge. His recent temperance, however, notwithstanding that it extended over so many years, was not enough to save him from the consequences of his former habits.

Five years before his admission he began to have œdematous swelling of his legs, but this did not last long: it was probably owing to some temporary defective action of the skin. His next symptom, which commenced six or eight months before he was admitted, was frequent micturition at night: he was compelled to get out of bed six or seven times of a night. This was probably due partly to an irritable bladder, and partly to the quantity of urine secreted. He next became affected with head symptoms: he suffered from violent pain in the vertex, which continued ever

after unabated. Soon after this his sight began to get dim, the little and ring fingers of his left hand became benumbed, and he felt very drowsy and unable to work.

The report goes on to describe him as "a heavy-looking man, thin and pallid; the complexion peculiarly sallow, as if not only the red particles of the blood were deficient, but also their hæmatine altered; frequently staring with a vacant gaze, answering questions very slowly, and appearing as if he felt it irksome to be made to reply otherwise than in monosyllables; moving his limbs about very slowly, and apparently with effort; pupils equal, contracting sluggishly."

Now, a patient coming to you with such a catalogue of symptoms as this, you would be tempted to set the case down as one of disease of the brain, in which the primary mischief is seated in that organ; and, if you made no further inquiry, you would immediately direct the whole force of your treatment to the head. Indeed, some years ago, this is what all practitioners would probably have done; for, in the state of knowledge then, it could not have occurred to them what else they should do: but we now know that such symptoms as I have already detailed to you, referable as they certainly are to the organs within the head, ought, nevertheless, to direct our attention to other organs likewise—namely, to the kidneys, and more especially if, in addition, the

patient has a pale sallow complexion, such as was very well marked in Jeffreys. Then, turning our attention in this direction, what information did we get in the present case? Why, we found that the quantity of urine was considerable, and that its specific gravity was very low; that it was acid, contained albumen, and that, on being allowed to stand, it threw down a scanty precipitate, which was found to consist of epithelium, of transparent casts of uriniferous tubes, and of a few cells containing a considerable quantity of fat.

Here, then, we get a satisfactory explanation of the head symptoms—a true and sufficient cause for them: we find them to be *secondary*, resulting from previously existing and primary disease of the kidney. It was evident that the general nutrition of our patient was considerably impaired; and, as the result of this impairment of nutrition in general, there was of course an impairment of the nutrition of the brain in particular: and moreover, although there was a considerable quantity of urine passed—for some days, indeed, the quantity of urine was so large, (as much as nine or even ten pints,) as to have led us to test for the presence of sugar—still it is probable, from its low specific gravity, that the due quantity of urea and other solids of the urine was not eliminated: and thus the blood, and the various organs, were poisoned at the same time that they were imperfectly nourished. And this twofold aberration

from the healthy standard—this actual poisoning, as well as imperfect nutrition of organs—this nourishment, by not only a poor, but by a poisoned blood, which inevitably results from chronic renal disease—explains how a train of symptoms similar to those above detailed may arise from a particular state of the kidneys. I do not say that these symptoms may not occur in cases where there is no kidney disease, but I do say that, in a considerable number of cases in which they are present, kidney disease will also be found.

The circulation was feeble, the pulse small and weak; but the heart's impulse was strong, and was felt over a greater surface than was natural. There was, however, no correspondence between the heart's force and that of the pulse, nor was there evidence of any valvular disease. At no time was there much dropsy. It never exceeded a moderate anasarca of the legs and a slight puffiness of the face.

This man continued in the hospital for four months, with scarcely any variation in his symptoms. He always attracted attention by his pale, sallow, and heavy look. He complained constantly of more or less headache, referred for the most part to the vertex, was always more or less lethargic, slept a great deal during the day, always heavily at night, but was frequently awaked up by frightful dreams. He was often sick, and vomited his meals. He had no paralytic symp-

tom, except a numbness of the left side of his face, and a similar numbness of the little and ring fingers of the left hand; which, however, sufficiently indicated that the impaired state of blood and of general nutrition was doing its work in the brain. Up to the day of his death he continued to pass pale urine in quantity varying from five to ten or eleven pints, never exceeding 1012 in specific gravity, and always containing a notable quantity of albumen, although not a very abundant precipitate in each portion examined.

For some days we compared the quantity of urine which he passed with the quantity of his drink, and found that they maintained a pretty close relation,—sometimes the latter exceeding the former, at other times the reverse; but in neither case was the difference more than from half a pint to a pint.

You will remember—those of you, at least, who are accustomed to accompany me through the wards of the hospital—that I had no difficulty in saying that the disease was in the kidney, and in indicating of what sort it was. I told you that the kidney was contracted, shrivelled up, from the atrophy and obliteration of numbers of the uriniferous tubes, altering the relations of the tubular and intertubular structures, creating congestion in patches and obliteration of some vessels, and interfering with the healthy formation of epithelium. This diagnosis rested upon the absence of more

decided symptoms of brain disease than the pain, the lethargy, and the numbness of one or two parts, and upon the known sufficiency of renal disease to disturb the nervous system in the manner and to the extent to which that disturbance existed in our patient. It was likewise indicated by the peculiar complexion of the patient (no hæmorrhage having taken place from any other organ than, perhaps, the kidney), by the presence of albumen in the urine, by the dropsy, and by the large quantity of urine passed.

This last symptom, with the low specific gravity of the urine, the comparatively small amount of albuminous precipitate, and the moderate extent of the dropsy, led me to infer that the disease was *contraction* of the kidney, due to a chronic degeneration of that organ. In cases of the enlarged kidney, which owes its increased size to the deposition of fat in the epithelium cells, or in that caused by the undue formation of epithelium distending the tubes, the urine is deficient in quantity; it has a smoky hue, often contains blood, the albuminous precipitate is very abundant, sometimes as abundant as in the serum of the blood, and the specific gravity is higher.

The diuresis which is often present in these cases, and which was so marked a feature of that which we are now considering, is explained by the condition to which the renal structures are reduced, as the result of chronic disease. The

tubes being stripped of epithelium, and consisting, in great part, solely of basement membrane, allow that to take place throughout their whole length, which in health would occur only at the Malpighian bodies—namely, the percolation of the aqueous part of the secretion. This great discharge of water was, however, a fortunate thing for our patient, for it enabled him to get rid of a much larger amount of solid matter than he would otherwise have been able to do. Had this free excretion of water been checked at any time, serious symptoms would no doubt have instantly shown themselves in epilepsy or profound coma. By Dr. Beale's analysis, it appeared, that shortly after his admission the urine, of which he then passed about five pints daily, contained twenty-two parts of solid matters in a thousand, and these consisted chiefly of *albumen* and *extractive matters*, whereas twelve or fourteen parts at least ought to have been urea. Even the increased flow of such urine as he was passing, would not have carried off enough of the organic and saline constituents to purify the blood. On the other hand, the constant drain of so much albumen, about 300 or 400 grains per diem, must have greatly injured the blood, as the nutrient fluid of the nervous system, by depriving it of so large a quantity of its chief staminal principle. And you can readily understand how even a slight check to the renal secretion would prove mischievous, by reducing

still further the small amount of excretion of the proper organic matters of the urine.

The prognosis of a case like this is always unfavourable. If the secretion be copious, the patient may linger on for a considerable time, as happened in the present instance; but if the secretion be materially checked, he will presently die. And although in our patient the flow experienced no diminution, he died suddenly, yet not unexpectedly, the manner in which he died differing somewhat from that of many other cases of the same disease. For some time his symptoms had been becoming aggravated; he had been more drowsy and lethargic, more doughy-looking and blanched, and the dropsy had gained on him, although not to any great extent. One night, without any special premonitory symptoms, he suddenly became comatose, breathed with stertor and puffing, his pulse became slow and languid, his respiration interrupted with increasing intervals, till it gradually and finally ceased. There were no convulsions. The time from the first access of the coma to his death was not more than a quarter of an hour.

The manner of death showed that it was owing to some change in the brain; and the short duration of the coma, and the speedy supervention of death, would go far to localize the lesion. Of this supposition the post-mortem examination was quite confirmatory: the following were the con-

ditions that it revealed:—On opening the cranium the membranes were found quite healthy; on removing the brain a clot was found in the fourth ventricle, which proceeded from the left lobe of the cerebellum, having broken down the substance of that centre to enter the ventricle. The blood had passed through the iter into the third ventricle; having filled which, it passed on either side, through the foramen of Monro, into the lateral ventricles, which it traversed, following the course of the choroid plexuses, and finally descending into the inferior cornua. The blood had coagulated into a firm clot, so that the whole could be taken out as a mould of the cavities and connecting passages. The choroid plexuses were lying on the inside of the clot, apparently healthy. The anterior cornua were filled with serum. The corpora striata and optic thalami, and the hemispheres of the brain, were healthy.

Now, in all cases where death supervenes very speedily on apoplexy, the clot is very likely to be found in a similar situation to this, involving, if not confined to, the mesocephale. Death is the more speedy the nearer it is to that important and central portion—that nucleus of the brain. In such cases death occurs much as it would do when an animal is pithed.

I may also refer you to a symptom which occurred in this case, and which is, I think, a very fatal one in apoplectic cases—namely, the flapping

of the cheeks in respiration. It is due to paralysis of both the buccinator muscles, and is the result of great compression of the brain, or of compression of it near the centre of respiration, which is also the seat of implantation of the facial and fifth nerves, upon which the action of these muscles depends.

The kidneys which I show you here were found in exactly the state which I described to you before the man died. Observe the surface of this kidney, how shrunk, granulated, and fissured it is, and on cutting it, how the organ is wasted, at the expense chiefly of the cortical substance. On a microscopical examination it was found that the tubes were stripped of their epithelium; in some parts the cells were filled with fat, but the chief feature as regards the epithelium was its scantiness and its imperfect organization. The thinness of the walls of the uriniferous tubes, as I have already explained, was favourable to the excretion of water, but not of organic matter, which is separated solely by the agency of the secreting epithelium; the former, holding albumen in solution, was, therefore, carried on with an energy proportional to its abundance, but the organic constituents of the urine would be secreted only in proportion to the quantity of epithelium existing in the kidney.

Coupling together the clinical history and the morbid anatomy of the case, there can be no doubt

that the state of brain was dependent on that of the kidney; and had it not been for what we may call the *accident* of the apoplexy, the patient might have lived on for some time longer, might have had several epileptic fits, and would probably have died in one of these, or from the exhaustion, which follows such attacks.

Taking together the state of the kidney and that of the cerebral functions, nothing can be clearer than this class of cases; but, putting the state of the kidney out of the question, nothing can be more obscure. There is no doubt that the kidney is the *primum movens:* the blood becomes poisoned; this poison interferes with perfect sanguification, a poor nutrient fluid is carried to the brain, which becomes, as a consequence, ill-nourished; add to this the probable actual presence of urea, and other solids of the urine, in the brain, and we have quite sufficient to account for all we have seen.

In the treatment of this case there was really little to be done. It is not improbable that by the frequent blistering of the neck and scalp, and other means of counter-irritation employed, such as the long issue in the scalp, we may have retarded the mischief within the cranium, and prolonged life somewhat; beyond this we cannot claim much credit for any benefit the patient may have derived. This is one of those maladies in which, the more we know as to its intrinsic nature, the more we feel the impotence of our art to

effect a cure. And so far, indeed, ignorance would have been bliss; in a certain sense, if it be not folly to be wise, our increased wisdom is attended, at least, with results less flattering and agreeable to our vanity. Still the knowledge of the disease, and a correct diagnosis, no doubt saved the patient from many blind and haphazard trials of remedies, which might have diminished his strength without any promise of good.

Another case that has recently come under our notice is, in one point of view, illustrative of what I have already said as regards the occurrence of cerebral symptoms without cerebral lesion, and it is also interesting on its own account.

Case XXV. "Maryanne Beasely, æt. 37, (vol. xxvi., p. 209,) native of London, where she has always lived; married, and has five children; of very intemperate habits, being much addicted to gin-drinking; in appearance thin and haggard, looking much older than her stated age; very weak and restless; her manner nervous and agitated; she has not slept for a considerable period; tongue somewhat furred, tremulous; pulse large, soft, not frequent." She had psoriasis on the back of both hands; for which malady, and for her general cachectic condition, she was sent to the hospital. She was admitted on the 1st of June, "and in the course of that night," the report of my clinical clerk Mr. (now Dr.) Hyde

Salter, goes on to state, "in no part of which had she slept, she was seized with furious delirium, and talked loudly and coarsely. By the negligence of the night-nurse, the house-physician was not called up: but on visiting her the next morning, she was found in a state quite resembling delirium tremens, suffering from various hallucinations of black-beetles, black men, and especially policemen, whom she appeared to hold in great horror. Stimulants and opium were prescribed, but without any immediate effect, and she became so violent that it was found necessary to remove her to a separate ward, and employ a strait-jacket."

Let me here remark, in passing, that in your practice the use of the strait-jacket should always be a last resource. If you cannot get a sufficient number of attendants, and there is danger of the patient doing himself injury, then it is absolutely necessary, and you have no choice; but it should be avoided if possible; it creates opposition, excites the patient, tends to prolong his delirium, and exhausts him. Remember, I do not say *never* use a strait-jacket, but make its *necessity* the rule for its use; for in many cases it would be highly dangerous, and even fatal to the patient's life, to dispense with it.

But to return to the history of the case. The delirium, notwithstanding the opium and stimulants, lasted till the night of the 4th, when she got some sleep, and from this time to the 8th she

rapidly improved, but on that day she began to squint. The urine was carefully examined, and found not to contain albumen. The report of the 8th says:—"The patient appears the same in every respect as when last reported, except that she has a slight but distinct internal strabismus, affecting principally the left eye: pupils rather dilated, but both exactly alike. She is much agitated at this uncomfortable symptom, but her agitation has not at all the appearance of delirium tremens. She states that when she looks at an object with both eyes, it looks indistinct and double, and she is totally unable to distinguish the distances and relations of surrounding objects: when, however, she covers up one eye, and it does not matter which, the vision is perfect."

She continued to improve, with the exception of the persistence of the strabismus, till the 12th, when she was taken back to the Augusta ward. Here she became delirious again, but the delirium was not violent; it was low and muttering: the squinting increased. The report of the 14th is as follows:—"The patient is a good deal weaker to-day: the squinting remains the same as when last reported. She is agitated, and rather more delirious, and the delirium is of a lower character, but not resembling delirium tremens in the least: pulse 110, weak: passes her motions under her. She has a good deal of bronchitis, diffused pretty generally over the chest; she coughs a good deal,

but does not spit up the mucus; and she passes her motions under her apparently from listlessness and inability of exertion:" in fact, she passed quickly into a typhoid state.

In the evening of the 14th she seemed to rally a little, but about one o'clock on the morning of the 15th a change for the worse took place; she became comatose, which state gradually increased, and she died about half-past seven in the morning.

My chief object in calling your attention to this case is to point out to you the great apparent want of correspondence between the state of the brain and the serious nervous symptoms under which the patient laboured. Nor, indeed, did the post-mortem examination bring to light such a diseased state of any other organ as sufficed to explain the fatal result.

There was considerable congestion of the lungs, and an abundant secretion of mucus in the bronchial tubes. The kidneys exhibited the appearance of passive congestion, but were otherwise healthy.

From the suddenness of the accession of the typhoid symptoms which came on at last, I was suspicious that our patient was suffering from purulent infection, that pus had formed somewhere, and had passed into the circulation. And this impression was the stronger from the resemblance of her symptoms to those of a woman who had been in the hospital some time ago. She was under treatment for chronic bronchitis, and

was going on tolerably well, when one day she became rapidly typhoid, with low delirium, her tongue furred and brown, and full quick pulse. This state continued for two days; then sudden coma came on, and she died. We found, on examining the body, an abscess in the septum of the heart: this had burst, and discharged its contents into the circulation. In many of these cases, if the patient does not die immediately, you get deposits in different parts of the body, particularly in the joints; but in such a case as I have related there was no time for such secondary abscesses. Among puerperal women we may often meet a case of this kind; a woman has been safely delivered, and everything seems to be getting on well; you visit her in the evening, and find a little uneasiness,—you order her bowels to be cleared out; the next day you find her sinking,—it may be, dying, and she may die within four-and-twenty hours of the first accession of the unfavourable symptoms. If you examine the state of the veins of the uterus, you will find the explanation—the woman had uterine phlebitis, and pus in her blood: these are cases that occur in the practice of every man.

But in this case we were not able to discover any source of purulent infection. Here is the brain: you perceive that it is decidedly paler than natural, and that those red points, which are produced by cutting across vessels, on slicing the white sub-

stance of the hemispheres, are perhaps too numerous and too large. With these slight exceptions the brain and its membranes seem perfectly healthy in their structure: there are no unnatural appearances; there is no evidence of any distinct lesion. And this is the point that I am most anxious to impress upon you,—that the delirium does not necessarily connect itself with any actual disorganization of the brain or its membranes, nor does coma either; but that both these formidable states may take place in a brain that shall reveal, on the minutest scrutiny, no appreciable aberration from the natural standard.

Now let us ask, of what did this woman die? I have already told you of my suspicion regarding purulent infection; but this must be abandoned, from our having failed to discover any purulent formation. I incline very much to think that the patient was not benefited by having been moved from one ward to another soon after she had been brought into the hospital.

She had been in a very exhausted state during her delirium: from this she had recovered to a great extent, and then she was moved up several flights of stairs to another ward. The fatigue consequent upon this, added to the attack of bronchitis, must have tended greatly to produce the typhoid state in which she died.

I have met with more than one instance of bad consequences following upon the removal of a

patient in delirium, or just recovered from it, prematurely. About two years ago a man was admitted here for epileptic delirium. Finding that his delirium was very noisy, and disturbed the other patients, I had him placed in a separate ward, where he recovered from his delirium. It was found necessary to move him upstairs, and shortly afterwards he became delirious again, and died comatose.

I am satisfied, from these and other cases, that there is nothing respecting which we ought to be more cautious than as to moving patients either in or just recovered from delirium; even to move them from one room to another on the same floor is dangerous, still more moving to any distance, or to another floor. Let us take this case as a warning of the necessity of great caution and circumspection before we sanction the removal of a patient under such circumstances.

LECTURE IX.

On Hemiplegia.

I PROPOSE, in the present and two or three subsequent lectures, to make some remarks on the clinical history, pathology, and treatment of that form of paralysis which, as affecting one half of the body, is usually called Hemiplegia. The affection is a very common one—we are rarely without two or three examples of it in the hospital; and you may often see persons labouring under it, walking in the streets with characteristic gait. Persons attacked with what is commonly called a paralytic stroke, are generally seized with this form of palsy.

Now, let me first describe to you the precise features of Hemiplegia, and the manner in which it differs from other forms of palsy, so that you may easily recognise it when you see it. You have an excellent example of it in the case of John Scott, in Fisk ward, and you may compare my description with the actual condition of that patient.

The term hemiplegia denotes a palsy stroke, affecting either half of the body; the parts actually involved are the upper and lower extremity, the

muscles of mastication, including the buccinator, and also of the tongue on one side. This must be distinguished from paraplegia, which means paralysis of the lower half of the body, in which both legs, and perhaps some of the muscles of the bladder and rectum, are paralysed.

You may have hemiplegia either *complete* or *incomplete,* as regards motor power, there being also great variety as to the affection of sentient power. In the complete paralysis, the upper and lower extremity on one side exhibit a complete loss of the power of motion, the face is very much affected and the tongue also.

When a patient is seized with this palsy, if he be standing or sitting, he falls, even though he retain his consciousness perfectly, because the power of maintaining his equilibrium is destroyed by the failure of the antagonizing muscles of one half of the body. On coming to such a case, you find the patient lying on his back, with total inability to move either the arm or leg; both of which lie, as if lifeless, by his side. Sometimes all power of motion is destroyed, and no movement can be excited either by any effort of the will or by any degree of external stimulation. In other cases, a very slight stimulus, such as tickling with the finger, or with the point or feather of a pen, or the application of a hot spoon to the sole of the foot, will, to the great surprise of the patient, produce active movements.

It is a point deserving of your notice, that very often either the movements thus excited, or the stimulation applied, although in itself not severe, will cause the patient a good deal of pain. It has often struck me that there is in these cases an irritable state of the sentient nerves, and of the centre of sensation, and that both the stimulus applied, and the jerking action of the muscles, create a good deal of pain. The patients frequently call out lustily from the pain thus produced, and express a great dread of, and dislike to, the repetition of the experiment.

You will also observe, that these excited motions, now so well known under the name of *reflex actions*, occur almost exclusively in the lower extremity. If you stimulate the palm of the hand, or pinch the skin of the forearm, you cannot produce movements such as occur in the lower extremity, nor, indeed, as a general rule, any movements at all, unless it be that when a patient has acquired some grasping power in the hand, tickling its palm, while he is attempting to grasp, will sometimes increase the power with which he performs that action.

There is, however, a curious and very interesting involuntary movement, which you will sometimes witness in hemiplegic cases. It occurs simultaneously with yawning, and less frequently with the actions consequent on emotion, surprise, joy or pleasure, or grief, as in laughter or crying. I may here mention that yawning is a very frequent,

sometimes a troublesome, and not always a favourable symptom after an attack of hemiplegia. It is more frequent in proportion as the shock is severe, but it seems to come on as the first effects of the shock are declining.

You will now proceed to examine the face. In slight cases this will not be at all affected, and the palsy will be confined to the limbs; sometimes, on the contrary, it will be the part first and alone paralysed. It is very necessary that you should be well impressed with the characters of the facial palsy which accompanies hemiplegia, and that you should be able readily to distinguish it from that which arises from affection of the portio dura. Place the patient well opposite you, and observe the conditions of the face, as it is perfectly quiescent. You will perceive that the paralysed cheek hangs, and that the angle of the mouth on that side is lower than its fellow; the cheek is more or less loose and flaccid, in proportion as the paralysis is more or less perfect. If now you ask him to smile or speak, the want of equilibrium of the face becomes very apparent. The healthy muscles being relieved of the antagonism of the paralysed ones, contract to a much greater extent than is natural, while the palsied cheek remains quiescent, or allows itself to be drawn slightly towards the mesial line. This exaggerated action of one cheek attracts very much the attention of the patient's friends, or himself when he looks in

the glass; and they are apt to refer all the mischief to the really sound cheek. "His face," the patient's friends will tell you, "is all drawn on one side;" and, they will hardly believe, when you assure them that the drawn side is all sound, and that its being drawn is merely the result of the want of a resisting power on the opposite side. But, with all this, the patient can shut both eyes well, and open them; and he can move the cheek, so far as can be effected by the zygomatic muscles, and he makes a very fair attempt at pursing up the mouth for whistling, this last act, however, being rather impaired by the intimate connexion of the buccinator with the orbicular muscle of the mouth. The muscles of mastication on the paralysed side act with less power, although it seldom happens in hemiplegia that their power is completely destroyed. Doubtless, much of their action is reflex, and this explains the fact, that while the buccinator muscle is very much paralysed, and even wasted, the masseter and other masticatory muscles retain a considerable amount of power.

Your anatomical knowledge will explain all this to you; the fifth nerve is more or less involved in or influenced by the paralysing lesion. In proportion to the extent to which it shares the depressing influence will be the number of muscles engaged, and the degree of their loss of power. If the nerve be immediately involved in the lesion, then the face-palsy is at its highest point. Some

of you may remember a striking instance of this in the case of a man named Coulson, subject to epileptic fits, who was a frequent visitor to the hospital, and was for some time a standing example of very complete palsy of the fifth nerve. In this man the face wore the same aspect as in hemiplegia, but, in addition, there was remarkable wasting of the temporal and masseter muscles, and a complete hollow in the regions which they occupied.

The facial nerve, or portio dura, is not generally touched by the paralysing lesion in hemiplegia. Hence you find the orbicularis palpebrarum and the superficial face muscles unscathed. In a few cases, indeed, it has appeared to me that these muscles were weakened, as if the nerve participated slightly in the shock; but complete palsy of the nerve rarely takes place, I would almost say never, unless the paralysing lesion is situated near its implantation. (Lect. III.)

In some cases the third nerve is paralysed, and this is shown by dropping of the upper eyelid and an inability to elevate it, and by squinting of the eyeball outwards, and more or less dilatation of the pupil. Not uncommonly palsy of the third nerve is a precursor of the hemiplegic attack, and its occurrence should always excite your fears of approaching more extensive mischief. Still it is remarkable that this nerve so often escapes in hemiplegia; how many hemiplegics do we not see

who have no trace of affection of the third nerve! Indeed, sometimes this will occur: a person will be seized with palsy of the third nerve and of no other; he will get well of this, and remain well some time; he will then be seized with hemiplegia, the third nerve escaping any fresh attack.

You now ask the patient to put out his tongue, and you observe a very characteristic phenomenon. The tongue is protruded with a more or less distinct deviation to the paralysed side. This is clearly owing to the impairment of the equilibrium of the protruding forces. Those on the right being paralysed or weakened, those on the left prevail, and push the tongue to the opposite side, that is, to the right. Very often the tongue-muscles are only weakened by the paralysing lesion, and the patient, on directing his attention specially to it, can protrude the tongue quite straight. I need scarcely tell you that this tongue-palsy is due to the ninth nerve being influenced by the paralysing lesion; the very same phenomenon may be caused by a local lesion affecting the nerve only.

In a physiological point of view, it is a very interesting inquiry how it happens that the fifth and the ninth nerves are so frequently—nay, almost universally—paralysed in hemiplegia, while the portio dura escapes. I cannot enter upon this point now, and must be content with remarking, that the discussion involves some highly

interesting questions connected with the mechanism of cerebro-spinal actions.

The palsy of the face, and that of the tongue, conjointly, give rise to the imperfection of articulation often present in hemiplegia. The patient speaks thick, and is especially indistinct in the pronunciation of labials and dentals, giving a guttural character to all his words. But sometimes the power of speech is wholly destroyed, even in cases where these nerves have suffered but little or not at all, or the power of utterance is limited to "yes" and "no," or either of these monosyllables; and this is a sign of very unfavourable portent, as denoting, with the other symptoms, extensive lesion of brain, superficial as well as deep.

It is curious how rarely it happens that the muscles of the trunk, as the intercostals, or the abdominal muscles, are involved in the hemiplegic paralysis. It must be an extensive cerebral lesion which will paralyse these muscles. There is, however, a spinal hemiplegia of which this palsy is a prominent feature.

Deglutition is also sometimes impaired, the vagus or glosso-pharyngeal being probably affected; but this is not a very frequent symptom, and, when present to any great extent, it denotes a serious and extensive lesion of the brain.

In grave lesions, especially when of the inflammatory kind, the sphincter ani muscle is paralysed.

This symptom, which augurs most unfavourably, is, happily, of only rare occurrence.

Such being the phenomena in hemiplegic palsy, let me say a word or two touching the way in which such an affection may be produced. You know that paralysis may be caused by any lesion which interrupts the continuity of a nerve or set of nerves, and which interferes with the due connexion between these nerves and the centre of volition; or by lesion of the centre of volition itself. Thus, then, you may have hemiplegia dependent on peripheral affection of the nerves, the morbid process spreading from periphery to centre—this is a rare and an incomplete form of hemiplegia,—or you may have it caused by a lesion in some part of the brain or spinal cord. If the lesion be situated within the cranium, above the point of decussation of the pyramidal columns of the medulla oblongata, the palsy will be on the side of the body opposite to the lesion: this is the most common form of hemiplegia. If it be seated in the spinal cord, below the decussation, the palsy will be on the same side of the body as the lesion; but in such a case, which is very rare, the phenomena present certain very essential points of difference from cerebral hemiplegia.

Looking at the matter, now, in a clinical point of view, I may state to you that the following forms or varieties of hemiplegia will occur to you

in practice, of most of which I hope to bring before you cases in illustration. First, and most commonly, you have the typical hemiplegia of diseased brain, that is, a brain affected with some distinct and special lesion, such as an apoplectic clot, a softening involving a considerable portion of the centre of volition, or a tumour in this centre, or compressing it. With this you may contrast the rare but not less certain spinal hemiplegia caused by a lesion involving one half of the spinal cord, just below the decussation of the pyramids. Thirdly, you may have hemiplegia consequent upon an epileptic attack, in which the paralysing lesion is generally transient, and the palsy remains only a few hours, or at most a few days, after the epileptic seizure. From this close connexion between the paralysis and epileptic fit, I prefer to mark this form of hemiplegia (although it may strictly be classed with the cerebral hemiplegia) as *epileptic hemiplegia*. Fourthly, you may have hemiplegia following, and sometimes, although rarely, preceding chorea—*choreic hemiplegia*. Fifthly, you meet with a peculiar and less perfect form of hemiplegia in hysterical women—the *hysterical hemiplegia ;* and in nervous, hypochondriacal men I have seen an analogous form brought on under the influence of strong emotion. Lastly, you have a form which, from its mode of access, creeping as it were from periphery to centre, you may call *peripheral hemiplegia*.

Now, in all these forms of hemiplegia the paralysis is prominently a paralysis of motion. This will occur in various degrees, from a slight awkwardness in the movements, to a completely paralysed condition, in which the patient does not possess the slightest power over the muscles of the limb. The extent to which sensation is impaired bears no constant relation to the degree of motor paralysis. You may have complete paralysis of motion with a sound or even an exalted state of sensation. In general, however, sensation is more or less impaired. In estimating this point, you must not be content with simply pinching up the skin of the patient, for there are many instances in which the sensibility is deadened, but in which it might escape our notice, unless we employed a more accurate method of investigation. The method I adopt is similar to that employed many years ago by Weber in comparing the sensibility of the surface in different parts of the body. It consists in ascertaining how near two sharp points of a pair of compasses may be approximated, and yet be distinctly felt as two points by the patient. Upon comparing the impression thus produced upon the sound limb with that on the paralysed limb, an idea and a definite expression of the extent to which sensation is involved may be arrived at. On the paralysed side the two points will be considered by the patient as but one, while on the opposite limb he will be able to distinguish clearly that there are two.

I propose in this and the subsequent lectures to illustrate these forms of hemiplegia by cases that have fallen under my notice; and I shall first speak of those cases which are distinctly caused by some special lesion of the brain.

I have many times had occasion to direct your attention to the condition of the muscles of the paralysed limbs in cases of cerebral hemiplegia. This point has not hitherto received the attention it deserves, and many otherwise interesting narratives of cases are comparatively of little value, from the omission of all mention of this symptom. Now I hope, as we go on, to show you that from the state of the muscles of the palsied limbs, especially of the upper extremity, as being nearest to the seat of the lesion, you may draw inferences as to the nature of the lesion which will afford important aid for diagnosis and prognosis; and I propose to make this the basis for an arrangement of cases of hemiplegia, which I trust you may find useful in practice.

Looking, then, to the state of the muscles of the palsied limbs, I arrange cases of cerebral hemiplegia in three classes:

The first class consists of those cases in which the muscles of the paralytic limbs are completely relaxed. The limbs are loose and flaccid, and if you flex the forearm upon the arm, or the leg upon the thigh, you find no resistance or opposition to that movement. When you feel the muscles you find them lax and flabby, contrasting

more or less with the firmness and plumpness of those of the sound limbs, and they are more or less wasted according to the period of time which has elapsed since the paralytic seizure.

In the second class I place those cases in which the paralysed muscles exhibit a certain amount of rigidity, *which rigidity has existed from the moment of or soon after the attack.* This rigidity varies in degree from an increased plumpness of the biceps of the arm and the hamstring muscles in the thigh, and a resistance on the part of these muscles to the extension of the forearm or leg, up to a contraction almost tetanic. The nutrition of the muscles in cases of this class is not materially weakened at first, and the wasting is consequently either *nil*, or to a very trifling extent. If, however, the palsy persist, the muscles waste, although not so fast as in the first class of cases.

In the third class, we find cases with rigid muscles likewise. In these cases the rigidity is a late phenomenon. It does not occur for some time after the paralytic seizure. The cases of the first class often pass into this. The wasted and relaxed muscles after some time gradually acquire more or less of tension; they become shortened, and appear like tight cords stretched between their origin and insertion. The tension is most manifest in the flexor muscles, and the limbs assume the state of more or less flexion, especially the upper extremity. The forearm becomes strongly

contracted on the arm, and the fingers flexed into the palm of the hand, which is liable to be irritated by the growth of the nails.

In all cases of cerebral hemiplegia, I advise you to pay minute attention to the investigation of the arterial system, and also of the heart. In old persons, or those somewhat advanced in life, we often find in the state of the radial or temporal artery a clue to the condition of the arterial system in general, and of the arteries of the brain in particular. In feeling the pulse you should roll the artery beneath your finger, and examine in this way as long a portion of it as you can get at. If the artery be diseased you will find a thickened state of its wall, and sometimes you will be able to detect distinct deposits in it, which now and then will be hard and resisting, owing to their admixture with earthy matter. You must be careful likewise to examine and compare the arteries of both sides, when you will often find corresponding states, and that the deposits exist more or less symmetrically. And this should confirm your suspicions, that the diseased state is not limited to the radial or temporal arteries, but exists pretty extensively throughout the arterial tree.

Your conclusion respecting the morbid state of the arteries will receive further confirmation if, on examining the heart, you obtain evidence of its being in a state of hypertrophy, for a morbid

state of the arteries is a fruitful source of hypertrophy of the heart. But, indeed, a disease of the heart of any kind, in advanced life, is very liable to be accompanied with a more or less diseased state of arteries.

Recently the observations made by Dr. Kirkes in this country, and by Virchow in Germany, have rendered it probable that disease of the brain, capable of producing hemiplegia, may sometimes be caused by the obstruction of a principal cerebral artery by a plug of fibrine detached from an excrescence on one of the aortic or other valves of the heart, the result of former endocarditis. You should, therefore, in examining the heart in young persons, look out for evidence of valvular disease caused by endocardial deposits.

Nevertheless, I must confess that I am not convinced that in cases such as Virchow and Kirkes refer to, the stoppage of the arterial circulation is always caused by a plug accidentally brought from a distant part of the circulation. I should be more disposed to refer it to a coagulum formed in the artery, promoted by an altered nutrition of its wall—*arteritis,* if you choose so to call it—and connected with a rheumatic or other morbid state of blood.*

That a softened state of brain—the state called

* See an interesting case recorded by Dr. Gibbon in the Transactions of the Pathological Society, Vol. V. p. 11.

white softening—follows the retardation and diminution of the cerebral circulation by diseased arteries, or its complete stoppage by a plugged artery, is now as well proved as any fact in pathology. To what extent the softening may go without producing paralysis, and whether, indeed, sometimes the mere shock of the sudden cutting off of a certain quantity of blood from a portion of the brain may not act of itself, without any softening of brain-tissue at all, these questions have yet to be decided. Neither has it been made plain, how the paralysis comes on so suddenly as it almost always does. Some would say it is because the stoppage of the blood-supply is sudden. But in many instances of softening from ill-nourished brain, the failure of the blood-supply is a very gradual process, and in some, in which the stream of blood is suddenly checked, paralysis does not come on for some time afterwards. Take, for example, some of the cases in which a ligature has been passed round the carotid artery. In Sir A. Cooper's well-known case, the palsy did not appear for seven days after the operation, and in one of Mr. Vincent's cases paralysis of sensation came on twenty-four hours after the operation, and four days afterwards paralysis of motion; and in my case of dissecting aneurism the paralysis did not ensue upon the plugging of the artery for three days.

I have been myself in the habit of attributing

the sudden occurrence of the palsy to the rupture or rapid deliquescence of fibres which had been already softened, but not sufficiently so as to interrupt their powers as conductors of the nervous force. When hæmorrhage has taken place into the substance of the brain, the effusion of blood is sufficient to tear across some fibres and compress others. But when there has been no hæmorrhage, the simple melting down of a portion of the nerve-fibres (promoted possibly by the passage through them of the nervous force, just as the platinum wire will be consumed under the transit of the galvanic current) will produce a solution of continuity, and stop the propagation of the nervous force. Such a view as this serves to explain the recovery of the palsy, on the supposition of the restoration of the normal nutrition of the nerve-fibres and the reunion of those which had given way.

I need scarcely remark that, reasonable as this view is, and sufficient as it is to account for the phenomena in many of the cases, it is very difficult of demonstrative proof. Nevertheless, I am not without hope that diligent microscopic investigation may yet demonstrate that such a solution of continuity of fibres does take place.

Let me now proceed to the first of the three forms of the hemiplegia of brain-lesion which I have described—namely, that with relaxed and flaccid muscles.

This form of hemiplegia occurs in two ways,—first, without loss of consciousness; and secondly, along with more or less of coma.

The first variety, for the sake of marking it distinctly, you may call *simple hemiplegia*. The patient is suddenly or rapidly seized with loss of power of one side without any comatose condition whatever.

Case XXVI. (vol. xxxvii., p. 194.) An excellent example of this form of hemiplegia—*the simple hemiplegia*—is to be found in a patient now in the hospital, James Scott, æt. 53, in Fisk ward, who has been subject to attacks of gout. His fingers exhibit deposits of urate of soda about the joints. He has had no other serious illness. The lung-sounds are healthy; there is a feeble but distinct mitral systolic bellows-sound, best heard at the apex of the heart.

This patient was admitted on the 29th of November, 1852, and the following history was obtained by my clinical clerk, Mr. Liveing:—Ten days since, while at work, he felt suddenly ill, was obliged to sit down, and discovered that he was unable to use his right side, but he did not lose his senses at all.

On his admission his condition was as follows—considerable palsy of the right side of the face; articulation slightly affected. Tongue deviates to the right side. He cannot move his right arm at

all: he is also paralysed in the right leg, but can move it very slightly. Sensation somewhat impaired in the arm. The pain produced by pinching is considerable in the leg, and less than might be expected in the arm, of the paralysed side. There is marked reflex action on tickling the sole of the right foot. The muscles of the paralysed limbs are perfectly relaxed, and quite free from any indication of rigidity. The arteries do not feel sound, being rather thickened. Pulse, 79. There is no evidence of disease of the kidney: urine natural.

Now I shall best explain to you the view I take of this case, by reading to you the diagnosis which I drew up after I had seen him, and which was recorded by Mr. Liveing in the case-book.

"Diagnosis.—Dr. Todd believes that there is, in this case, white softening, most probably of the left corpus striatum, and, perhaps, also of some of the fibres of the hemisphere round about it. He conjectures that it may have resulted from obstruction of a cerebral artery (perhaps from gouty deposits), giving rise to a gradual softening, and ultimately to rupture or rapid deliquescence of fibres, which is the immediate cause of the sudden paralysis. The obstruction may be temporary only, and if the force of the circulation be sufficient to overcome it, or a free collateral circulation be established, the nutrition of the brain may go on as before, and the patient recover."

The patient was on the whole in so fair a state of general nutrition, that I was led to augur favourably respecting him. Still I might have added, that the failure to overcome the arterial obstruction or to establish a collateral circulation would have resulted in confirmed palsy with relaxed muscles, or, after a time, in slow contraction of the paralysed and wasted muscles, and a state of permanent flexion of the forearm and hand, and some stiffness of the leg.

The progress of this case was as follows:—

On the 3rd of November, five days from his admission and fifteen from the attack, the following note was made in the case-book: "Much better altogether; he can move the fingers of the right hand, and flex the elbow-joint of the same side. Sensation is also improved; he can distinguish two points on the back of the hand at an inch and a quarter apart; feels pain now readily on pinching. The right leg is also better—he can move it better—no pain in it, and the pain on pinching is less than it was: passed three pints and a half of urine in the last twenty-four hours, specific gravity 1017: right pupil larger than the left."

On the 7th, all the symptoms had improved excepting the paralysis of the arm; on the 9th, the palsy of the face was much better, and he was able to protrude his tongue nearly straight. The inequality of the pupils was less obvious; on the

16th, he had regained strength so much that he was able to walk with assistance on each side.

This man improved steadily and uniformly up to the beginning of February, when his farther progress was checked by an attack of the gout in his left knee; this yielded in a few days to treatment, and he remained in the hospital till the 22nd of February, when he was discharged, having recovered the power of the paralysed side almost completely, nearly three months from the attack.

It was particularly deserving of notice, that in this case the muscular power was restored without any permanent state of rigidity or contraction of any muscle or set of muscles of either limb. In consequence of this favourable restoration of the paralysed parts, I would infer that a collateral circulation restored the nutrition of the softened fibres, and that whatever solution of continuity had taken place then, a process of union (by the first intention, as one would say, of external parts) had been established.

In some cases the improvement in the softened part of the brain is, apparently, accompanied with a complicated process of cicatrization of the brain-substance, and connected with that is a change in the paralysed muscles, which, without regaining their normal state of nutrition, become rigid, and throw the limbs into a more or less permanent state of flexion. Others, again, of which Case XXII., related in the seventh lecture, is an example,

make very little progress towards recovery, the limbs remaining in the relaxed and flaccid state which they had assumed at first. I shall, in a future lecture, call your attention to these modes of termination of hemiplegic cases of this class.

If this man's (Scott) arteries are diseased, as is most probably the case, it is not unlikely that sooner or later he will have a second attack, and that then the softening and the solution of continuity of fibres, and it may be hæmorrhage, will take place in the right hemisphere of the brain.

I have already detailed a striking example of the tendency of both sides of the brain to become diseased, causing double apoplexy, (vide Lect. VI. Case XX.) I will now give you a more perfect example, not only in illustration of the tendency of the brain symptoms to recur by disease of the opposite hemisphere, but also showing how complete may be the recovery from the effects of the first attack of simple softening, and of simple hemiplegia consequent thereon.

Case XXVII. Mr. J. R., æt. 75, a gentleman who had filled with distinction for many years the office of Actuary in a leading Insurance office in London; he was a short, stout, plethoric man, of sufficiently active habits both of mind and body. One night, in the beginning of the month of December, 1850, having been previously in very good health, although for some time gradually

failing in general power, on leaving the drawing-room to go to bed, he found, after taking hold of a bedroom candlestick with the left hand, that he was unable to lift the candlestick, and the arm immediately fell powerless to the side, completely paralysed. His consciousness was not at all impaired; so perfect indeed was it, and so much did he retain his presence of mind, that he at once directed his servant to fetch from his library the volume of the Encyclopædia containing the article Paralysis, that he might ascertain whether he was attacked with that affection. His usual medical attendant, Dr. Woolley, of Brompton, was sent for, and on his arrival the leg had become quite paralysed, as well as the face and tongue. He was bled moderately, and treated chiefly with aperients.

I saw him two days after the attack, and found complete paralysis of the left arm and leg, with a perfectly flaccid and relaxed state of the muscles. The face and tongue palsy were quite complete. Sensibility was only very slightly affected, and there was no pain in the head. The heart's action was good—rather quick—and a slight thickness was perceptible in the radial artery. Speech was but very slightly impaired, and the intelligence was perfect.

I continued to attend this gentleman, in conjunction with Dr. Woolley, for many months. In the month of May of the following year he had

regained his power to such an extent, that he was able to go out and enjoy walking exercise; and, ultimately, towards the close of the summer of 1851, he so completely regained his power, that no difference could be observed in the movement of the left leg from that of the right, excepting, perhaps, a slight stiffness, and he could use his arm and hand freely, and could grasp with full force. There was but one defect in the hand, namely, a slight stiffness and semi-flexed state of the second and third fingers, which interfered with the perfect use of his fork at table. This symptom was never removed.

I had frequent opportunities of seeing this gentleman during the remainder of 1851, and throughout 1852, during nearly the whole of which time he retained the full power over his left side. At the end of the former year he began to suffer from an irritable and inflamed bladder, which secreted pus and phosphate of lime in considerable quantities, and he would, under these circumstances, pass highly alkaline urine. This state was kept in check effectually by large doses of the dilute nitric acid.

So he went on till the middle of 1852, when along with his bladder symptoms he manifested a good deal of debility, and an extreme fidgetiness and restlessness, and a certain amount of peevishness of temper; his memory likewise rather failed, and he could not walk as he had previously done,

being very easily fatigued. I had referred these symptoms to a gradually and slowly progressive softening of one or both hemispheres of the brain; and I had prepared his friends for some sudden change, either a fresh attack of paralysis, such as he had previously, or one accompanied with apoplectic effusion, which might speedily terminate his life. And I also intimated that the attack would not improbably affect the left side of the brain, as previously the right.

One Tuesday night, towards the end of December, 1852, having passed the day in his usual health, and been very cheerful, he was suddenly seized, soon after going to bed, with paralysis of the *right* side, and comatose symptoms very rapidly supervened. He quickly fell into very profound coma, with stertor and flapping of the cheeks, and died after some hours in the course of the night.

The examination of the brain was conducted by my friend, Mr. Dixon, surgeon to the Royal London Ophthalmic Hospital. The right hemisphere, including the corpus striatum and optic thalamus, which must have been the seat of the original disease, appeared healthy—we failed to detect any mark of previous lesion; but it is not impossible that such mark may have escaped our search, as it was conducted under very unfavourable circumstances, in a small room, on a dark day in December. The left lateral ventricle was full of blood,

which had flowed freely into the right ventricle, the third ventricle, and through the iter into the fourth ventricle. The left corpus striatum and optic thalamus were completely swept away by the gush of blood, and the white substance of the hemisphere outside them was torn up by a large coagulum, the nervous matter beyond being evidently softened, and breaking down readily under a slight stream of water. The arteries were extensively diseased, and there were numerous fatty deposits in the walls of the smaller vessels.

A third case may be here quoted, showing complete recovery after a first attack, and partial recovery after a second in which the paralysis affected the opposite side.

Case XXVIII. Mary Anne Godfrey, æt. 29, a housemaid, of temperate habits, and without any history of syphilis, was admitted into the hospital on May 21st, 1850, with hemiplegia of the *left* side (Vols. xxxi. p. 147, and xlix. p. 116). The paralysis of motion was not quite complete, as she was able to move the fingers of her left hand *very slightly,* but the sensibility of the palsied limbs was almost entirely suspended; there was ptosis of the left upper lid, the tongue when protruded diverged towards the paralysed side, and her articulation was very imperfect. The sounds of the heart were normal, and the urine, upon examination, was found to be healthy. The account

which this patient gave of herself was this:—Her health had been tolerably good up to December, 1849, when she had an attack of rheumatism, for which she was treated in a London hospital; and in January, 1850, she had a fit (the first in her life), which lasted two hours, and which, from her own description of it, appears to have been of an epileptic character, but out of which she came with the full use of all her limbs. In the beginning of the following March she had a second epileptic seizure, which also lasted about two hours, at the end of which time, when she came to herself, she found that her left arm was paralysed, and her face drawn to one side; the palsy, however, did not last longer than half-an-hour. Three weeks subsequently to this she had a third and similar attack, which was also followed by transient paralysis of the left side. From this time she continued without the occurrence of any fresh fit up to May 17th (1850), on the morning of which day, after having slept very heavily the preceding night, she awoke with complete hemiplegia of the left side, and in this state she remained until she was brought to the hospital four days subsequently. She was at once subjected to a supporting plan of treatment, and, under the administration of quinine, iron, and an occasional purgative, together with a nutritious diet, she gradually recovered the use of her limbs, and was discharged on July 4th (1850), *i. e.*, about six

weeks after her admission, perfectly free from every trace of paralysis.

[I regret to say, the condition of the paralysed muscles during this attack was not noted.]

After leaving the hospital, the health of this patient continued pretty good, with the exception of her being more or less liable to rheumatic pains, until the evening of November 6th, 1855, —a period of more than five years—when she suddenly fell down in a state of insensibility whilst walking in the street. She was taken home, and the following morning a medical man was sent for, who cupped her on the back of the neck. She continued, however, perfectly insensible until the night of the 7th, when she recovered her consciousness, but only to find herself hemiplegic on the *right* side; and on the 14th, the paralysis continuing, she was again brought to the hospital. On this occasion there was complete paralysis, both as regards *motion* and *sensation*, of the right arm and leg, and of the right side of the face and tongue; articulation was much impaired, and the muscles of the paralysed limbs were perfectly flaccid and relaxed, offering not the least resistance to the movements of flexion or extension. No reflex actions could be excited by tickling the sole of the *right* foot, nor by the application of a hot spoon to the same limb. A bellows-sound, accompanying the systole of the heart, was distinctly audible, and was

loudest over the apex of the organ.* The patient was again put upon a supporting plan of treatment, consisting in the exhibition of ammonia, chloric æther, and small quantities of brandy, along with a generous diet; and, for these, iron and quinine were after a time substituted. Under this course she gradually but slowly improved, and on February 28th, 1856, after a stay in the hospital of rather more than three months, she was again discharged, having completely recovered the sensibility of the paralysed parts, and having regained the power of voluntary motion in the lower extremity to such a degree as to be able to walk about the ward with the assistance of a nurse. The arm continued quite paralysed as to motion, with some degree of contraction of the flexor muscles of the fingers.

* This bellows-sound was observed during the patient's former sojourn in the hospital, and was probably due to an endocardial affection at the time of her rheumatic attack in 1849.

LECTURE X.

On Hemiplegia with Relaxed Muscles.

In my last lecture I introduced the subject of Hemiplegia to you, and enumerated the various forms in which (looking at it in a clinical point of view) the affection is apt to occur.

You will remember that I proposed to you two forms of the simple hemiplegia, or that with relaxed muscles. In both, the attack of paralysis is sudden, or at least very rapid; but in the first form it is not attended by any affection of consciousness, while in the second it is accompanied by a comatose condition which lasts a longer or shorter time.

Now the first of these forms of the simple hemiplegia I illustrated by the case of J. Scott, who had been in the hospital, and by another case which I attended in private. Both these cases are examples of the affection occurring in the advanced period of life, at which time, indeed, the attack is most apt to take place—because it is at that time that the arteries undergo the changes which render them less fit as carriers of blood to the brain, and also make them brittle.

You will meet with cases of this kind at the

earlier periods of life likewise; but at these ages the obstructed circulation will not arise so much from chronic disease of the bloodvessels as from a sudden arrest of blood in them, either by the formation of a plug in their interior, or by its detachment from some other part of the vascular system, and its impaction in one of the chief cerebral arteries.

The cases of this kind of hemiplegia recorded by Dr. Kirkes were aged respectively, 34, 24, and 24; and Dr. Burrows has also contributed a case at the very early age of 11, and another, most probably of the same nature, aged 19.* These communications are of very great interest and importance; and the proved existence of cases of this kind will of course lead every practitioner to investigate the condition of the heart, and to seek for evidence of a rheumatic diathesis, or of attacks of rheumatic fever in the antecedents.

The case of M. Godfrey, quoted in the last lecture, might be possibly referable to a lesion of this kind.

It is at the earlier periods of life that you will meet with, also, the cases of what I propose to call choreic hemiplegia, and also most of the cases of epileptic and hysterical hemiplegia. You will, therefore, bear in mind that in investigating cases of hemiplegia at the early ages, it will be necessary for

* Medical Times, 1853. No. 136.

you to determine whether they have any connexion with these states of constitution.

The pressure of a tumour so far impairs the nutrition of surrounding parts of the brain as to induce white-softening, and consequent paralysis or other symptoms.

By and by I shall have to detail to you the particulars of a case of hemiplegia in a young person of nineteen, in which the diagnosis was for some time obscure, but which was referable to the form of the malady now under our consideration.*

I must ask you to remember, that obstructed blood-vessels are not the only cause of the softening, upon which this form of paralysis depends. A poor or vitiated blood, excessive mental effort, grief, fright, an abnormal deposit, a severe epileptic fit, the syphilitic poison, may so impair the nutrition of the brain, as to cause softening and solution of continuity of brain-fibres, even although the arteries be healthy and pervious.

You will recollect that I insisted much upon the importance of noting carefully, in your records of cases of hemiplegic paralysis, the condition of the paralysed muscles; and that I proposed to make that the basis of my arrangement of such cases for clinical observation. This subject is so important, that I must beg of you to excuse my

* Vide infra, Case L.

referring to it again. In all cases of this description I would ask you to observe and note the following points: 1st, whether the muscles of the paralytic limbs are absolutely relaxed and flaccid, and offer no resistance to the extension or the flexion of the limb; 2ndly, whether, when you attempt to extend the forearm upon the arm, you experience resistance from the biceps muscle, or, if you flex the one upon the other, you find the triceps to cause resistance; and in the lower extremity, whether you encounter similar resistance from the hamstring muscles, or from the rectus femoris: 3rdly, whether the muscles are rigid, and maintain the limb in a more or less flexed state; and lastly, if rigid, to note the time at which the rigidity came on,—whether it came on simultaneously with, or soon after the palsy, the muscles being well nourished—or, at a remote period, with wasting of the muscles.

A very little reflection will show you the reasonableness of attaching importance to the state of the muscles in brain and spinal-cord affections. If you observe the results of experiments on animals recently dead, you will perceive in what an intimate relation the nervous and muscular forces stand to each other. You cannot touch a muscular nerve, or a part of a nervous centre in which muscular nerves are implanted, without affecting the muscles to which those nerves are distributed, however distant they may be. How instantly does

strychnine signalize its presence in the blood, by the tetanic condition of the muscular system! This is not because strychnine has any special affinity for the muscles, but because it has the property of exalting the nervous force, of exciting the polarity of the nervous centres, and this in its turn can excite the muscular force. If, then, the state of the muscular force is an index to the condition of the nervous force and of the nervous centres in poisoning by strychnine, why may we not use it to enable us to form some estimate of the state of the nervous force in paralytic cases? And may we not with justice affirm, that when the muscles of a paralysed limb are in a state of rigid and tetanic contraction, the nervous centre at or about the lesion is in a very different state, *quoad* its power of generating the nervous force, from that in which it is, when the muscles are lax, flaccid, and entirely inactive?

No observer, so far as I know, has paid attention to the condition of the muscles, exactly in the way that I propose. Lallemand, and more recently Durand-Fardel, seem to me to have done most in this way, and to have had a clearer appreciation of the importance of looking to the state of the muscles, than any other observers. But they have not sufficiently noted the occurrence of rigidity as an early and a late phenomenon.

The lesion which gives rise to the paralysis in these cases, is "white-softening," *i. e.*, softening

of the brain substance without any discoloration; the cerebral matter becoming so soft that a gentle stream of water poured on it from a height breaks it up (which has no such effect on a healthy brain), and in some cases, indeed, becoming diffluent, so as to assume a creamy consistence.

The suddenness of the attack of paralysis is due (as I suppose) to rupture of the softened fibres. It must, I think, be admitted, that fibres, wanting in their normal consistence, may, nevertheless, propagate the nervous force, and that a softened brain may more or less perfectly minister to volition or sensation. Persons, whose brains are wholly or partially in this condition, often experience uneasy sensations in the limbs, which subsequently become affected, such as neuralgic pains, numbness, coldness, &c. By and by fibres give way, sometimes all at once, often gradually one after another, and the paralysis ensues with proportionate rapidity.

The second variety of the first class of hemiplegia, is characterised by the suddenness of the attack, and by more or less of coma soon after, or simultaneously with the paralysis. This form of hemiplegia, to which I wish to direct your attention to-day, is more common than the simple variety of which I have hitherto spoken, and it is also of a much more serious nature, as it often quickly terminates in death. It comes on in this way:—A man is walking about, or following his

usual occupation, or doing any particular action, when his arm or leg suddenly becomes paralysed; at the same time he experiences a certain amount of shock, and soon the other limb on the same side also becomes affected. In the course of an hour or two, he falls into a sleepy state, which passes into coma, the breathing becoming stertorous, the face drawn to one side, and the flaccid paralysed cheek flapping to and fro, which is always a symptom of very bad omen. Sometimes, however, the coma and palsy come on simultaneously; at other times a fit of epileptic character occurs, from which the patient recovers paralysed, and then passes into coma.

In illustration of this form of hemiplegia, especially as regards its mode of invasion, I shall refer you to the following case very lately in the hospital.

Case XXIX. Elizabeth Harvey, æt. 54, who was admitted into Lonsdale ward, on May 3rd, 1853 (the notes of whose case have been kept by my clinical clerk, Mr. Plowman, in the most praiseworthy manner). (Vol. xxxix., p. 258.) She was in her usual state of health, until three days before her admission into the hospital, when she experienced a shock, on recovery from which she found that her left arm and hand hung useless by her side. She soon passed into a state of stupor, her breathing became stertorous, and she was

brought into the hospital in an imperfectly comatose condition. By talking loudly to her, we could get her to answer questions, and in this manner we elicited the account which I have just narrated. There was complete hemiplegia on the left side, the muscles of the limbs being all completely relaxed, with the exception of the biceps of the arm, which, on extending the forearm on the arm, became firm and resisting. In this last respect, the case scarcely comes under the class of hemiplegia with relaxed muscles; but I quote it as illustrating that form in all the main points; by and by, I shall refer to this particular symptom. Our patient complained a good deal of headache; the left side of the face was paralysed, the mouth being drawn to the right; the tongue, when protruded, had a marked divergence towards the paralysed side; there was some difficulty of swallowing; and thus early in the case there was slight puffing of the left cheek, and she passed her urine and evacuations under her, as if from paralysis of the sphincters. The following day, May 4th, she became perfectly comatose, and the left cheek flapped to and fro with each expiration. This phenomenon of flapping of the cheek, as I have already remarked, indicates a very perfect paralysis of the buccinator muscle, and is usually associated with an extensive paralysing lesion. The pupils were dilated, the right being larger than the left; and the pulse was slow, being on

the 3rd, 56, and on the 4th, 52, while the respirations were eighteen in a minute.*

Let me detail to you another case in which the muscles were completely relaxed, not even the biceps becoming rigid on extension of the forearm.

Case XXX. Jane Power, æt. 63, admitted September 18th (vol. xii., p. 118). On the 16th of September, she complained of pain in the right side, the nature of which could not be ascertained. This pain passed off, and on the 17th, she was as well as usual. On the evening of the 18th, she suddenly complained of being very ill, and put her hand to her right side. It was found that the whole of that side was completely paralysed, and that it was cold. She did not become unconscious at once. She was immediately brought to the hospital.

On admission she was found in a state of stupor, and took no notice of those around her, but when roused would answer questions apparently correctly. Her breathing was somewhat stertorous; deglutition rather difficult. She put out her tongue when asked to do so, and it deviated to the right side. The pupils were much contracted. Pulse 100, very compressible. The paralysed limbs were quite relaxed; when the arm

* See the conclusion of this case at page 203.

was lifted up and let go, it fell upon the bed as if dead. All her evacuations were passed in bed, the sphincters being probably paralysed.

On the 20th and 21st, she seemed to have rallied a little, and answered questions more readily, and swallowed better; the pupils were less contracted. On the 22nd, without apparent cause, she became again less conscious; pulse 120; extremities cold. She lingered on till the 28th, the paralysis remaining unchanged, and the circulation failing, the lungs also becoming much congested. She died at seven o'clock A.M., on the 28th.

The lesion in this case was extensive white softening of the middle of the left hemisphere above the roof of the lateral ventricle, in the centre of which a clot as large as a small walnut had formed. A small opening had been formed into the ventricle. The cerebral substance around the clot was much softened, and had some small extravasations in it. There is unfortunately no mention made in the record of the case of the condition of the arteries, so that we are left in the dark as to the cause of the white softening.

The case of Ware, detailed in Lecture V., p. 91, is a good illustration of this form of hemiplegia with relaxed muscles. This patient, as you will remember, was suddenly seized with paralysis at night, so that it was impossible to say whether his consciousness was, or was not, affected at the

moment of the seizure. When he awoke, he found the right side of his body paralysed, and soon after he fell into a comatose state, which lasted for a considerable time; on recovering from the coma, he was still hemiplegic, and the paralysed muscles were completely relaxed. In this state, he was brought into the hospital; he died of erysipelas of the head and face, and thus an opportunity was afforded of ascertaining the condition of brain associated with the relaxed state of the paralysed muscles. Upon examining the brain of this man after death, the left corpus striatum was found to contain a small cavity, which was full of fluid; the cerebral substance immediately around this cavity, was completely softened; there appeared to have been no attempt at the healing process; and, with the exception of the softening surrounding the cavity, the rest of the corpus striatum was in a healthy condition.

Case XXXI. There is at present a man in Rose ward, of the name of Shea, who well illustrates this form of paralysis and its progress (vol. xxvi., p. 98). This man was admitted into the hospital for the first time on April 4th, 1849, (four years ago,) and his history is this:—On April 2nd, 1849, while going down to Sydenham by rail, he had a fit in the carriage, and when the door was opened, he was found lying on the floor of the carriage, with paralysis of the right side of

his body. A medical man, who was called in to see him, bled him to ten ounces, and ordered him some aperient medicines; and two days after the attack, he was brought to the hospital. He then had hemiplegia of the right side with complete relaxation of all the paralysed muscles, but the biceps contracted on making extension of the fore-arm; his face was drawn to the left side, the tongue, when protruded, diverged towards the right, and his speech was a good deal affected. After a time he recovered to a slight degree the power of the right arm; since then he has been more than once an inmate of the hospital, and he is now again a patient; I shall presently have to refer to him more particularly in illustration of the changes which such cases undergo in the course of time (p. 208).

Having detailed to you these cases illustrative of this form of hemiplegia, I shall now speak of the different modes in which it may terminate.

This variety of hemiplegia may terminate in one of four ways:—1st, it may end in death, and that, too, pretty rapidly; 2nd, it may terminate in permanent paralysis, the paralysed muscles remaining relaxed; 3rd, partial recovery of the paralysis may be attained, the paralysed muscles becoming wasted, and passing into a state of rigidity; and, lastly, there may be complete recovery of the paralysis.

When the patient dies, death results from the exhaustion consequent upon the shock and the suspension of function of a portion of the cerebro-spinal centre. The rapidity of the fatal event will be determined mainly by the extent of previous brain disease, or by the quantity of blood effused, or by the part of the brain affected—sometimes by the intensity of the shock. With regard to the part of the brain affected, I think it is quite certain, that when the apoplectic clot compresses directly the pons Varolii, or the medulla oblongata, or parts immediately connected with them, death will take place much sooner than when any other part of the brain is involved. Moreover, in cases in which the brain-lesion has, of itself, exercised a very depressing effect upon the powers of life, it is by no means to be overlooked, that the duration of life may be materially influenced by the treatment adopted, at the time of, or soon after the attack. Some will recover, to a certain extent, the immediate effects of the brain-lesion, but will succumb sooner or later by the failure of general nutrition.

In the sequel of the case of Elizabeth Harvey, part of which I have read to you, (Case XXIX.,) you have a good illustration of a common mode of fatal termination of hemiplegic cases, when effusion of blood has occurred.

On the 4th of May she passed into a state of complete coma, the palsy remaining as before;

the pupils becoming more dilated, the right appearing rather the larger; the pulse as low as 58. On the 5th the comatose state continued, and she was evidently sinking. The pulse had fallen to 50, and it was interesting to notice that the heart's action was 90—a striking proof how much the force of the heart was weakened, for nearly each alternate systole was so weak as to be incapable of generating a pulse at the wrist. She died this night at ten o'clock, six days after the first attack of palsy.

Before examining the body after death, I ventured to make the following diagnosis:—I stated that I believed the symptoms were due, primarily, and essentially, to white-softening, consequent upon disease of the cerebral blood-vessels; that hæmorrhage had taken place, with more or less laceration of brain-substance. The latter point rested chiefly upon the rigidity observed in the biceps muscle of the arm whenever the forearm was extended on it. From the flapping of the cheek, I thought that the hæmorrhage was probably considerable; and that it was not unlikely that blood had been effused into the lateral ventricles.

Upon examining the brain the following were the appearances noticed:—The membranes appeared healthy; there were two or three enlarged Pacchionian bodies: there was no subarachnoid effusion. The brain-tissue generally appeared

somewhat soft, and the convolutions of the anterior and middle lobes of the right hemisphere (the paralysis was on the left side) were greatly wanting in consistence, and readily gave way under a fine and gentle stream of water carefully poured on them. The brain did not appear to be at all congested, and there was no effusion into either of the lateral ventricles. On slicing the hemispheres, a small clot of blood, about an inch in diameter, was found in the right corpus striatum, and the whole of this body, and the surface of the optic thalamus of the same side, were also extremely soft, and were readily broken down by allowing a gentle stream of water to fall on them. The left hemisphere appeared to be healthy, as did also the cerebellum. We then proceeded to examine into the cause of the white-softening and apoplexy, and we found by microscopic examination evidence of most extensive disease of the small arteries and capillaries of the brain. The vessels of the softened portion of the corpus striatum immediately surrounding the clot were thickly studded with oil globules, which, in some situations, were aggregated into dark masses, so large as here and there almost to fill up the vessels. The minutest capillaries, as well as the larger arteries, exhibited these deposits, and few could be discovered free from them. Acetic acid rendered the walls of the vessels and the globules clearer, but did not bring out the muscle-cells, whence we infer that, in

part at least, the fatty deposits resulted from the degeneration of these fibres. No effervescence occurred on the addition of this reagent, nor did any of the globules appear dissolved by it. When, however, a drop of ether was added to one of the vessels, the globules were dissolved, and a greasy stain remained on the glass on the evaporation of the ether. Thus we had sufficient chemical evidence of the fatty nature of these particles. Many of the vessels of the vesicular matter of the convolutions exhibited this same fatty disease, only in a less degree.

Our diagnosis, then, appeared to be correct in all the main points. It was wrong, however, in the estimate of the size of the clot, and in stating that the hæmorrhage had extended into the lateral ventricles. This error might, I think, have been avoided, had I allowed sufficiently for the length of time (six days), which elapsed from the seizure. The great extent of the softening sufficiently accounted for the comatose state, which ensued so early, and could scarcely have been produced by so small a clot, in the midst of healthy brain. The brain had already, before the rupture of the blood-vessels, been much diseased, and had no power to rally after the severe shock which was inflicted upon it: but death will not occur, under these circumstances, so quickly as when an extensive hæmorrhage takes place into the lateral ventricles.

I may refer you to the cases of Thurston and Regan, both described in Lect. VII., for instances of the speedy death of patients where large clots are formed in connexion with disease of the same kind as that observed in this patient. And, in the case of Catherine Williams, detailed in Lect. II. and III., you will find a well-marked instance of death under this form of hemiplegia, where there was no clot at all, and in which, if any blood had been effused, it must have been in very small quantity.

Other patients die from the effects of bed-sores, and with indications of the failure of general nutrition, promoted, no doubt, by the depressed state of the whole nervous system. The case of J. R., quoted in my last lecture, is an example of this.

The second mode of termination may be illustrated by the case of Ware (p. 91), in whom death did not ensue on lesion of the brain, for he recovered from the loss of consciousness and other immediate effects of the attack, and, indeed, from the paralysis to some degree, (being just able to move the fingers slightly), but on erysipelas. I believe, however, that if he had survived the erysipelas, he would never have completely recovered the power of his limbs. Around the excavation which was found in the corpus striatum, there was no indication of any contraction or cicatrization, and the absence of this accounted for the

condition of the affected muscles. There was just enough of the corpus striatum left to enable the patient to recover some power over the paralysed parts, but not sufficient to restore them to their normal state. You will remember that as much as six months had elapsed from the paralytic seizure to his death, affording ample time for a cicatrizing process to have been established.

An illustration of the third mode of termination is to be found in the case of Shea, now in Rose ward. (Case XXXI.) This man recovered from the immediate effects of the attack, but the paralysis remains; and all his symptoms favour the opinion that in his case there has been rupture of some blood-vessels, with the formation of an apoplectic clot in, or very close to, the left corpus striatum, and hence the very complete paralysis of the right side which at first took place. After a time he recovered some power over the leg and arm, but the muscles of the paralysed limbs have gradually assumed a remarkably rigid condition, the arm being drawn to the side, the forearm flexed on the arm, the wrist bent on the forearm, and the fingers closed on the palm. When an attempt is made to extend the arm, great resistance is offered by the flexor muscles, particularly the biceps, which feel hard and rigid, and as if in a somewhat tetanoid state. At the same time, all these muscles have wasted very much, and are much smaller than those of the other side. This

represents a maximum of this condition; but, in many instances, contraction is limited to a few muscles only, or to the biceps, or the flexors of the fingers, or to portions of them, so that a patient will recover with permanent flexion of one or two fingers. I shall recur to this point in my next lecture.

Lastly, and least frequently, recovery may take place without any rigidity or change in the muscles, other than a gradual restoration to their normal condition. Of these, I related to you two striking instances in my last lecture in the cases of J. Scott, and J. R. In J. R.'s case a slight stiffness of two fingers remained, and also some rigidity of the semi-tendinosus muscle.

When recovery takes place, you should observe the order in which the palsied parts become restored. The leg, face, and tongue are generally the first to recover; sometimes the face and tongue are first, at other times the leg; almost invariably the arm, as if it were most severely struck, from being nearest to the seat of lesion, requires the longest time to shake off the paralysing influence of the seizure. But even this sometimes begins to recover before the leg.

Such being the Clinical History of the first form of Hemiplegia, in its two varieties, what can we say as to its Pathology?

It may, I apprehend, be pretty confidently affirmed respecting the first variety, namely, that

in which there is simply paralysis without coma, occurring *suddenly* or *with great rapidity*, that it is always dependent on softening, without or with a clot; that the softening is of the colourless kind, and that when a clot exists, it is so small as not to exercise pressure on neighbouring healthy parts of the brain; and, respecting the second variety, in which more or less of coma prevails, that it is also dependent on softening of the same nature, without or with a clot. When without a clot, the softening is of considerable extent, and may perhaps be associated with some previous morbid deposit.* When with a clot, which is the more common case, this is either of large size, or it is situated in some part where it can compress central and important parts of the brain.

The evidence now accumulated respecting the lesions which give rise to these two forms of hemiplegia, indicate, I think, very distinctly that they result from defective circulation through the brain, and enfeebled nutrition of the cerebral matter. In some instances, actual obstruction of important arterial channels can be shown; in others, there is a marked degeneracy of a large portion of the arterial and capillary system, which may have preceded or gone on simultaneously with the cerebral degeneration. In all cases the

* In illustration of this form of softening in connexion with strumous deposit, the reader is referred to Case L., Lecture xiii.

cerebral disease reaches such an extent, that the vesicular matter imperfectly generates the nervous force, and the fibrous matter becomes a bad conductor of it, or even a non-conductor, or its continuity is interrupted, and so its power of conduction is rendered, in the one case, physiologically, in the other, mechanically impossible. And, if the softening of brain have been of sufficient duration, there will be found in it the large vesicular bodies, containing fatty particles in a state of minute division, which indicate a further degeneracy of the brain tissue, or an attempt at a reparative process.*

There are some other points which I must notice, such as the differential diagnosis and the prognosis of this form of hemiplegia; but these I would rather postpone until I have spoken of the other forms.

Let me, now, ask your attention to a few remarks upon the treatment of cases of this description. You are called to a patient who has become the subject of sudden paralysis and relaxation of the muscles of one half of the body: what can be done? We will suppose that you are satisfied that the attack results from the changes connected with white softening and diseased arteries. You will, naturally, reflect upon the changes that have

* Vide p. 132.

been going on in the brain. The nerve matter has been undergoing a change which, either by alteration of structure, or by a solution of continuity, or by both, renders it incapable of propagating the nervous force. One or more blood-vessels have given way; or, if not, their rupture is imminent. You will ask yourself, what can I do to prevent this threatening hæmorrhage, or to check it if it have begun, or to restore the conducting power of the nerve-fibre?

I believe that the most important end for the practitioner to aim at, in the early treatment of these cases, is to keep down the frequency and force of the heart's action, by means not depressing to the vital powers. For this purpose the strict maintenance of the horizontal posture is of the highest moment; and when the patient is conscious, it is most desirable that the mind should be tranquillized by every means. It will, of course, likewise be necessary to remove all local impediments to the easy flow of the circulating fluid; and it is as well that the head should be slightly raised, sufficiently to prevent gravitation favouring the escape of blood from the ruptured vessels, but not so as to create any impediment to the flow, which might embarrass the action of the heart.

To remove any distant source of irritation from the alimentary canal, which may be operating injuriously on the brain, the bowels should be

cleared; and in order that there may be as little effort as possible on the part of the patient in the expulsion of the contents of the bowels, it is expedient that this should be done by enema; but if this fail, and the vital power of the patient do not forbid it, you may give croton oil, a drop or two of which, placed on the tongue, will operate freely; or calomel, in powder, to the extent of five or ten grains, which may be similarly administered.

I would advise you to limit the further administration of drugs, to giving some slight corrective, as an alkali,—ammonia being, on the whole, the most appropriate,—unless, indeed, you find the patient in an extremely prostrate condition, when it will be necessary to combine with it the cautious exhibition of other stimulants and restoratives, as chloric ether, brandy, &c.

The question of bleeding will arise; and, under the popular notion, that all head attacks are accompanied and caused by the rush of blood to the head, you will be pressed to have recourse to this expedient.

There are three objects to be attained by bleeding: first, to diminish an undue amount of blood in the head; secondly, to check hæmorrhage or to prevent it; and lastly, to quiet the heart's action.

Let me briefly point out to you the circumstances under which bleeding is inadmissible.

If the patient be cold, and collapsed, it is clear you should not take blood; nor should you have recourse to this practice if the heart's action be very feeble or intermittent; nor if there be an anæmic state; nor if the patient be of very advanced age; nor if the evidence of extensive disease of the arterial system, or of the heart, leave no doubt on that head; nor would it be desirable to bleed if it were clear that already a large amount of hæmorrhage had taken place into the brain.

Should none of these objections exist, then you will have to consider whether any or all of the indications above named need to be fulfilled, and whether bleeding (local or general) promises to fulfil them.

As to the first indication—namely, the diminution of an undue amount of blood in the brain, I think modern investigation of the actual state of that organ clearly points out, that the brain is not in a hyperæmic state, in the cases in which the form of hemiplegia under discussion is likely to occur.

Will taking blood check or prevent hæmorrhage? The sudden or rapid abstraction of a moderate quantity of blood, either from the arm or temple, or by skilful cupping from the nape of the neck may, I can conceive, check hæmorrhage: and with this object it is, sometimes, a very justifiable practice, but the quantity taken should not

be large. Now and then bleeding helps to diminish the frequency and force of the heart's action; but here, again, the quantity of blood withdrawn should be moderate, for the removal of much blood is apt to quicken the heart's action and render the blood poor.

I would have you to look upon this question, to bleed or not to bleed, as almost the most important one you will have to decide; and, judging from my own experience on this point, as well as from the results of the practice in a large number of cases collected from various sources, as I pointed out in a former lecture (Lect. vi., p. 117), I have come to the conclusion that, in cases of white softening, with or without hæmorrhage, you are less likely to err by omitting rather than by adopting the practice.

It sometimes happens that in these cases a rigidity of the muscles comes on very early, which indicates an inflammatory process going on around the clot, which may end in the formation of pus and abscess, and is to be combated by the use of mercury. But you must be careful to distinguish this from the muscular rigidity which is of late occurrence, and results from a restorative effort of nature; and with which it is, therefore, not desirable that you should interfere.

You will often be consulted as to "some expedient for promoting the restoration of the paralysed limbs to their normal condition." To this

question, after having given a fair trial to the various means which have been proposed for this purpose, I must reply, that I know of nothing, which more decidedly benefits the paralysed limbs than a regulated system of exercise; active, when the patient is capable of it; passive, if otherwise. As to the use of electricity, which is now much in vogue, or the employment of strychnia, which has been strongly recommended, I feel satisfied, as the result of a large experience, that the former requires to be used with much caution, and that the latter is very apt to do mischief, and never does good. I have seen cases in which, after the employment of electricity for some time, that agent has apparently brought on pain in the head, and has excited something like an inflammatory process in the brain. And strychnia is also apt to induce an analogous condition of brain, and to increase the rigidity of the paralysed muscles.

Some good may occasionally be effected by the use of frictions, or cold water, or shampooing, all of which tend to improve the general nutrition of the nerves and muscles.

In my next lecture, I propose to speak of that form of hemiplegia, which is associated with a more or less rigid condition of the paralysed muscles.

LECTURE XI.

On Hemiplegia with Rigid Muscles.

You will remember that the first kind of hemiplegia of which I spoke, was that which is accompanied with a relaxed state of the paralysed muscles, and that I described two forms of it—the one in which there is sudden paralysis, with a relaxed condition of the paralysed muscles, without loss of consciousness; the other in which these phenomena are associated with loss of consciousness, to a greater or less degree, at the moment of, or soon after, the palsy-stroke.

I mentioned, also, that the second kind of hemiplegia is where the muscles of the paralysed limbs are rigid, and where this rigidity comes on simultaneously with the paralysis, or very soon after it. You must bear in mind that a distinctive feature of this hemiplegia is the very early period at which the muscles assume this rigid condition. This is the more important, inasmuch as we meet with another form of hemiplegia with rigid muscles, in which the stiffness gradually supervenes a *long time* after the paralytic seizure, and may succeed to the relaxed condition of the paralysed muscles.

My purpose, to-day, is to bring before you examples in illustration of the clinical history and morbid anatomy of hemiplegia, *with early rigidity of the paralysed muscles,* and to make some remarks on the pathology of this form of paralysis.

Now of this kind of hemiplegic paralysis, you will in practice meet with two varieties; the one, in which the rigidity of the paralysed muscles is very slight, and confined to one or two muscles; the other, in which it is considerable, and affecting all, or nearly all, the muscles. The former of these is very apt to occur in those cases of hemiplegia, in which most of the paralysed muscles are flaccid, one or two only being in a rigid condition; and, as illustrations of this form, I adduced cases in my last lecture, in which there was a greater or less impairment of consciousness—where a clot had been formed with laceration of brain-substance, and where hemiplegia resulted, accompanied with a flaccid condition of all the muscles of the paralysed arm and forearm, except the biceps; other cases will occur in which there will be slight rigidity, not only of the biceps but also of the triceps and the flexors of the fingers, and in a still less degree of the hamstring muscles and the biceps femoris. In many cases of this description, the rigidity of these muscles will not be apparent, unless they are thrown into action by exciting their antagonism. Thus, when you attempt to extend the forearm upon the arm, you

will find that the biceps will become more or less stiff and rigid, and resist the extension; and so also will the triceps resist flexion; and, in like manner, will extension of the fingers be resisted by the flexores digitorum. In general the actual assumption of the rigid state, or the tendency to assume it, is more marked in the flexor muscles than in the extensor, and in the upper than in the lower extremities. It likewise very rarely affects the muscles of the face, or any other paralysed muscles than those of the limbs; but sometimes the muscles of mastication are involved, as the patient, although insensible, will resist powerfully any attempt to open the mouth.

This condition, of slight and partial rigidity of muscles, is that of most frequent occurrence in the hemiplegia caused by an apoplectic clot. My idea as to its cause is, that it depends upon a state of irritation, propagated from torn brain to the point of implantation of the nerves of the affected muscles. But, you will ask, why is it that in some cases of clot the hemiplegia will be accompanied with complete relaxation of muscles, while in other cases the rigidity of which I have spoken exists? The answer to this question is as follows: in the cases where there is no rigidity the clot lies in the midst of softened brain, and has not in any degree encroached upon sound brain; but when rigidity exists, the clot has extended beyond the bounds of the white softening,

and has torn up to a greater or less extent, or irritated, sound brain. I leave this explanation to be tested by further experience and observation.

I am anxious to relate to you one or two cases in addition to those which I have already detailed in my last lecture.

Case XXXII. Mrs. C., æt. 57, had been for some time in a depressed and nervous state, with failing memory. One day at noon, when walking into her drawing-room, she suddenly became paralysed on the right side, and would have fallen had not the servant caught her; she became speechless, and the only sign of consciousness she showed was evinced by her weeping at the sight of her daughters. She quickly passed into coma with collapse. I saw her soon after, with Mr. Dunn, of Norfolk-street.

We found her lying in a state of insensibility, from which, by very loud speaking, she could only be roused so as to open her eyes; she was breathing slowly, but without stertor. There was hemiplegia of the right side, with rigidity of the biceps, which resisted extension. She kept her mouth firmly closed, and when an attempt was made to open it, she resisted so powerfully that it was found impossible to introduce anything in the natural way. Two of her teeth on the right side were gone, and through the vacancy fluid could be introduced, which she swallowed with

facility. On introducing the pipe of an enema-syringe, it was found that the sphincter offered no resistance, and was paralysed. She quickly showed marked indications of collapse, on taking away a few ounces of blood by cupping. Reflex actions could be produced by tickling the sole of the right foot. The pupils were much contracted.

These symptoms continued till the morning of the third day, when the coma became profound, and she died on that day. On examination, it was found that the left hemisphere, just external to the corpus striatum, contained a large clot, the size of a bantam's egg, dark and firm. This clot had torn up the surrounding brain substance, and had excavated a cavity for itself; the cerebral substance around this clot did not seem to be particularly softened, but presented a very peculiar appearance, in consequence of being studded over by numerous small coagula (capillary apoplexy). The corpus striatum was firm, and seemed compressed by the clot. The optic thalamus was natural. The arteries of the brain, to their minutest ramifications, were studded with atheromatous deposits. The skull was very thick; in front seven-eighths of an inch.

It is most probable that in this case, owing to the disease of the arteries, there first occurred a softening of a considerable portion of the left hemisphere, just outside the corpus striatum. In this softened portion several small vessels gave

way, and a large hæmorrhage broke down the softened cerebral matter and encroached upon the healthy brain, compressing the corpus striatum.

Here is another instance of a fatal case of this form of hemiplegia :—

Case XXXIII. A man named Frost (vol. xxxi. B, p. 15,) a water gilder, of intemperate habits, was brought into the hospital in a state of coma. It was then found that his right side was paralysed ; the right side of the face seemed motionless, and the right arm also, which exhibited rigidity of the biceps and triceps muscles ; the leg was partially paralysed, and was frequently moved in a jerking manner. Pupils contracted. He was bathed in a profuse perspiration, and his heart was acting feebly ; pulse 80. No satisfactory history of his attack could be obtained, beyond that it had occurred suddenly, and that he was immediately brought to the hospital.

On the following day (Feb. 14th), he became more conscious, but still remained very stupid ; the right arm and leg paralysed, the arm being rigid, but only as regards the biceps and triceps ; the leg twitched convulsively now and then ; pupils contracted and equal ; deglutition not impaired. On the 21st, there was no other change than an improved state of consciousness, and a slight increase of power in the leg. On the 25th, he was attacked with severe diarrhœa, under which he sank rapidly.

The *post-mortem* examination revealed softening of the white substance of the left hemisphere, outside of and on a level with the optic thalamus. Within this the optic thalamus was broken down, to the extent of its external two thirds, by a clot of quite recent black blood, which encroached on the corpus striatum, having slightly torn and compressed it. The nervous matter immediately around the clot was discoloured by intermixed blood; that, external to it, was softened, and free from discoloration. In the latter the nerve-tubes were found to exhibit their natural structure, in the former there were numerous nerve-tubes, with blood corpuscles, and numerous vesicular bodies filled with highly refracting fatty particles.

Now, in this case, the existence of the compound cells in the softened brain substance immediately around the clot, seemed to me to indicate that a process of softening had been going on in the brain, anterior to the rupture of blood vessels. This rupture was followed by laceration and compression of parts immediately influencing muscles, as the corpus striatum and optic thalamus; and hence, not only was the lesion paralysing, but likewise, to some extent, irritative, giving rise to the rigid state of the biceps and triceps muscles.

A much more interesting form of the second kind of hemiplegia is that in which there is considerable rigidity of all the muscles of the arm and forearm; where the arm is kept at an angle with the trunk (and sometimes these patients hold

it across the chest), the forearm being flexed on the arm, and the fingers bent on the palm. In these cases the paralysed muscles appear to be firm and contracted, and sometimes in an almost tetanic state, and offer considerable resistance to extension or flexion, which frequently also excites a good deal of pain. When the rigidity is of this nature, the paralysis is generally not complete, a certain degree of the power of moving the whole limb or some part of it being still retained; and very frequently sensibility is affected, being sometimes obtuse, but oftener in an exalted condition, while it not uncommonly happens that reflex actions are also considerably exalted. In the latter cases, you will find the excitation of reflex action produce a considerable degree of pain. No doubt the same cause, which gives rise to the rigid condition of the paralysed muscles, contributes to produce the exaltation of the reflex actions. In hemiplegic paralysis reflex actions are usually most marked in the lower extremity; indeed, it is very rare to see them well developed in the upper; but in these cases the application of a stimulus to the palm will sometimes excite them in that limb, and in the lower extremity they will seldom fail to be produced by stimulating the sole of the foot, or pulling a hair of the leg.

This form of hemiplegia sometimes occurs in surgical practice, in consequence of a blow on the

head, with depression of bone, or from considerable hæmorrhage within the cranium, such as results from injury of the middle meningeal artery, or one or more of its branches. The hæmorrhage, thus produced, separates the dura mater from the adjacent bone, and exerts pressure on the corresponding surface of the hemisphere, and this, in turn, gives rise to paralysis of the opposite side of the body, accompanied, very often, with a rigid condition of the paralysed muscles. Indeed, when hemiplegia with a rigid state of the muscles supervenes soon upon some injury to the head, you may, almost always, make a certain diagnosis, that it is the result of irritant compression of the opposite hemisphere, by depressed bone or by hæmorrhage outside or inside the dura mater. A well-marked case of the kind occurred some time ago in Albert ward. I happened to come in, as the patient was admitted, and found him lying in a comatose state, with great rigidity of one side of the body, while the other was in its normally flaccid condition. This immediately directed my attention to the side of the head opposite the rigid muscles, and, after death, we found a large effusion of blood on that side, between the dura mater and the bone, the result of injury to the middle meningeal artery.

Sometimes inflammation of the pia mater or arachnoid causes an accumulation of puriform fluid in the sub-arachnoid or arachnoid spaces, which, by

compressing the corresponding surface of the hemisphere, gives rise to this form of paralysis on the opposite side of the body. A good example of this occurred in the following case :—

Case XXXIV. A woman named Wilson, æt. 35 (vol. xxxi. B, p. 51), was admitted with syphilis into the surgical wards, but symptoms very like those of typhus fever having manifested themselves, she was transferred into Lonsdale ward, where she came under my care. For a day or two, we were at a loss to determine, whether she was suffering from arachnitis or typhus fever; for, as you know, it is often a matter of great difficulty, and indeed impossible at first, to distinguish the effects of the typhus poison from those of meningeal inflammation, upon the nervous system. When she was admitted into our ward, on the 8th of March, her symptoms were as follows: a flushed face, hot and dry skin, tongue dry and coated with a thick black fur, sordes on the teeth, intense thirst. She lay constantly on her back; the bowels were very much relaxed, and her pulse was small and frequent, 120, and compressible. She had vomited two days before her admission into the medical wards, and had a shivering, and complained very much of headache, not referred to any particular spot. On the 9th there was a muttering delirium; she passed all her evacuations under her; the tongue was still very dry and

black, with sordes on the teeth; pulse 112. Next day, the 10th, much more stupor; decubitus on the back. A new symptom now presented itself; this was hemiplegia of the right side; the palsy of the right upper and lower extremities, especially of the former, was accompanied *by a rigidity of the muscles,* described in my notes of the case as "almost tetanic." The tongue was protruded with difficulty to the right side, the *left* pupil was fully dilated, and the right was also dilated, but less so than the other. She died on the 11th, and after death we found, that a copious effusion of pus had taken place in the arachnoid sac, over the surface of the left hemisphere of the brain, and that, being confined by certain adhesions, it had compressed and hollowed out a cavity on the corresponding surface of the convolutions. Lymph was effused over the whole surface of the hemisphere, inwards to the falx, and forwards to the fissura Sylvii.

This case well illustrates the mode of formation of this kind of hemiplegia, showing that it is due to a cause, which exercises at once a paralysing and irritating influence on the brain. This influence is propagated to the spinal cord, and through the nerves, implanted in that portion of the nervous centre, to the muscles of the paralysed limbs, in which it excites a state of contraction. The effect is analogous to that produced by the continued action of the electro-magnetic

machine, which you sometimes see in use in the hospital, and just as a rapid succession of electric shocks may maintain a constantly rigid condition of muscles, so may continual shocks of nervous force, due to irritative pressure, bring about a similar result.

An affection of the substance of the brain, of an irritative character, may give rise to this form of hemiplegia. Of this you will find a good instance, in a patient lately under our observation, of which Mr. Plowman has kept an excellent record.

Case XXXV. The subject of the case was Mary Reeves, a cook, forty-nine years of age. She appears to have freely availed herself of the opportunities which the kitchen afforded to indulge in intemperate habits. She had suffered, for some time before her admission, from severe pain in the right arm and leg, which her medical man told her was gout. One evening, while out, she was seized with pain in the right foot, which was so severe as to make her limp in her gait, and oblige her to obtain assistance to walk home. The next day, however, the pain was better, but a paralytic state of the right arm and leg had come on. Notwithstanding this, she was able to walk without assistance; but she dragged her leg, and the arm hung useless by her side. She now put herself under medical treatment, and her

health and strength improved, but the paralytic state remained the same.

On Monday, the 9th of November, she lost her way while endeavouring to walk to Chelsea, and had to come back a considerable distance, but did not seem the worse for the exertion. But on the following Saturday she became much worse, having had a fit as her friend supposed. She fell to the ground insensible, but was not convulsed, nor did she bite her tongue; she recovered from this quickly, but a similar attack recurred daily. During all this time she faltered very much in her speech, and exhibited a considerable amount of drowsiness. Her bowels had been much confined, on the Tuesday and Wednesday she had no evacuation, but on Thursday, the 18th, she began to pass everything under her in bed. The drowsiness and paralysis increased until her admission on November 20th, and when brought into the hospital she was in a semi-comatose state, would answer questions only when loudly spoken to, complaining of headache, chiefly frontal (from which she had suffered all along), and was paralysed on the right side of the body, the palsied muscles being extremely rigid. The arm was bent firmly across the chest, and she retained a little power of motion in it; reflex actions were very marked in the lower extremity; the tongue, when protruded, did not diverge particularly to either side; the left pupil was dilated; and there was

slight external strabismus, and also a slight amount of ptosis of the left lid. In this state she continued; and though various remedies were used, at first mercury and subsequently iodide of potassium, she gradually sunk, and died eighteen days after her admission.

Upon examining the body of this woman, we found the right hemisphere of the brain in a healthy condition. The surface of the left hemisphere was rather flattened, and its convolutions expanded. On slicing, it was found evidently much congested, as shown by the numerous bloody points from cut veins in the white substance, and more especially by the dark colour of the grey matter of the convolutions. The white matter of the anterior lobe of the left hemisphere, and the surface of the corpus callosum, were much softened, as was demonstrated by pouring a gentle stream of water upon it; and the softening extended from the convolutions to the corpus striatum, the anterior and inferior portions of which were completely broken down, leaving, however, many of its fibres intact and free. At the posterior part of the horizontal portion of the corpus striatum, there was a mass which presented the appearance, as far as regards colour and consistence, of a disintegrated blood-clot, consisting of a gelatinous looking substance, which resembled fibrine in an imperfectly coagulated state, together with some colouring matter, through which nu-

merous nerve-fibres were found passing, but no blood-globules. The lesion extended backwards to the corpora quadrigemina, and downwards to the inferior layer of the crus cerebri; and thus arriving at the base of the brain, it could be traced to the fissure of Sylvius, where a considerable induration was found. I am inclined to believe that the disease consisted primarily in this induration, and that the lesion gradually spreading upwards from this point finally involved the anterior lobe. The posterior portion of the upper surface of the mesocephale towards its continuation into the thalamus, and the posterior part of the thalamus itself, were in a very indurated condition.

Now, without attempting to determine, whether this extensive brain-lesion was inflammatory or not (which from the want of a minute microscopical examination is impossible), there can be little doubt, that there was quite enough alteration in the induration and the extensive white softening, to excite irritation and to cause paralysis. And it was to this *irritative*, as well as *paralysing* lesion, that the rigid state of the muscles of the paralysed limbs was to be attributed. It is probable, as I have already said, that the starting point of the disease was at the indurated portions. They, no doubt, had much to do with the early attacks of epileptic character, with the faltering speech, and the semi-comatose state. To these symptoms

were soon added the paralysis and the rigidity of muscles.

Rostan gives an interesting case of this form of hemiplegia, with a rigid state of the paralysed muscles, in which the symptoms depended on an exostosis, projecting from the parietal, and the petrous portion of the temporal bones, and compressing the cerebellum, so as to form a deep hollow on its surface, thus causing paralysis with rigidity of the opposite side of the body.

In Lallemand's work, there are the narratives of several cases of this form of hemiplegia with early rigidity, in all of which the brain lesion was of an irritative, or of an inflammatory character. I must limit myself to quoting one case. It is the first of his second letter. A man, æt. seventy-six, was brought into the Hôtel-Dieu, on the 1st of April. He had been found lying insensible on the floor of his room. The limbs of the left side had lost sensation and motion, but they were semi-flexed, stiff and contracted, especially when an attempt was made to extend them. He regained his intelligence slightly, but remained in a lethargic, almost comatose state, the palsy being unchanged, until the sixth, when he died at noon. A very thin layer of lymph covered the arachnoid on the right hemisphere. There was extensive softening of the middle and posterior lobes, and the softened matter had the colour and consistence of pus, and in the upper part of the posterior lobe

a small deposit of pus was found. Towards the inferior part of the middle lobe, the softened grey substance had a brownish colour from the infiltration of a small quantity of blood, and in this situation the vessels were dilated and filled with blood.*

The cases which I have detailed to you, and it would not be difficult to multiply them, will, I think, sufficiently illustrate the mode in which the hemiplegia with early rigidity is apt to come on. Let me point out to you, that it is not an inflammatory state of brain only, which may excite this rigid palsy, but one of irritation, whether inflammatory or non-inflammatory. By an irritated

* In the Proceedings of the Pathological Society of London for 1851-52, there is an account of a very well-observed case, in which red softening appears to have supervened upon white softening. The hemiplegia was on the left side, and the muscles of the arm, at first, seemed to have been flaccid, and to have subsequently become rigid. The case is recorded by Drs. Sibson and Handfield Jones.

Dr. Bennett has put on record a case in which there was twitching of the muscles of the right arm, followed by numbness of the fingers of the right hand, and diminished power of the arm on the subsidence of the twitchings. The right leg was only benumbed. This state was followed some weeks after by an attack of coma, with rigid flexion of the right arm, and great resistance when an effort was made to extend it. The lesion consisted of inflammatory induration surrounded by softening of a portion of the left cerebral hemisphere.—*Clin. Lectures*, No. IV. p. 152.

state I mean, one of exalted polarity of the nervous tissue, or as the physical philosophers would say, a state of high tension. Inflammation may exercise as depressing an influence as atrophy; in other words, it may as effectually destroy the conducting power of the nerve fibres or the generating power of the vesicular structure, as if the elements of those two kinds of nerve-matter were wasted or ruptured. And as Lallemand has remarked, an early stage of the inflammatory process may be irritative and paralysing; a later stage paralysing simply.

You may fairly ask, is a paralysing lesion compatible with an irritative one? To this I think an affirmative answer may be readily given. A state of irritation interferes with the due conducting power of the nervous fibres, and with their prompt obedience to the influence of the will. The physical nervous action refuses either wholly or in part to obey the mental influence which usually controls and directs it; the disturbed nutrition of the nervous matter prevents the ready development of those physical changes in the nerve cells and nerve fibres, without which the will cannot be freely obeyed. Moreover, in many cases, the lesion consists at once of ruptured fibre and compressed brain substance.

In further illustration of this form of hemiplegia, let me call your attention to a symptom which often occurs in cases of cerebral disease.

It is this, that not only is hemiplegic paralysis present in a more or less complete form, with or without rigidity, but also frequently a convulsive condition, with or without loss of consciousness. Now and then, the patient will be seized with more or less active clonic, and sometimes choreic convulsions of the paralysed limbs, and occasionally these convulsions will pass into the complete epileptic paroxysm. The convulsive attacks to which I allude, which may be appropriately called *epileptiform*, last sometimes as long as twenty minutes or half an hour, leaving the patient greatly exhausted, and the limb paralysed.

One of the best marked examples of this kind, which I have seen, was in the case of a little child, whom I attended some years ago along with Mr. Dunn. In this case these convulsive movements were an early and a prominent symptom. The convulsions were of the clonic kind, and sometimes while the arm was jerked convulsively, the leg was affected with tonic spasm. The left side of the body was affected with the convulsions, and it was found, after death, that a considerable deposit of tubercle had taken place in the pia mater of the convolutions of the right hemisphere, around which an extensive inflammatory softening had occurred.*

* See Mr. Dunn's account of this case, in *Med.-Chir. Trans.*, Vol. XXV.

Another interesting example of similar convulsions some of you may have witnessed in the hospital, in a man, who had often been the subject of our clinical observation. His name was Beglin; he was first admitted with a remarkable spasmodic rigidity of the right forearm, which was cured by iodide of potassium. He was a man of intemperate habits, and after every bout of drinking he would be liable to attacks of convulsions, sometimes with complete, at other times with partial insensibility, the convulsions always affecting the right side. For these attacks he frequently applied at the hospital, and was an inmate of it on more than one occasion. With a rare gratitude for the interest which I had taken in his case, he left instructions before his death that I should be invited to examine his head. We found over the left hemisphere of the brain a thickened state of the dura mater, and marks of old inflammation of the other membranes.*

I have seen similar hemiplegic convulsions in women during pregnancy and after parturition. In one of these cases, a lady whom I saw in October, 1852, with Mr. Muriel and Dr. Young of Kennington, there had been previous attacks of epilepsy. The second case was a lady whom I attended with Mr. Street and Mr. Hetley. This

* See the full particulars of this case in Lecture xvii., and the remarks in that lecture generally.

lady was a perfectly healthy person, and had gone through her confinement without the least untoward circumstance. Being one of those persons who get over confinements very readily, she was able to sit up at the end of a week. One day she seemed to have done too much; and that evening she was suddenly taken with a convulsive fit, which was strictly limited to one side of the body, and was accompanied with loss of consciousness. These convulsive attacks were repeated, and at such short intervals that it was evident, if something were not done to check them, she must soon sink exhausted. She had been bled early, and mercury was freely exhibited, but without affecting the severity or the frequency of the attacks. Under the use of chloroform the fits were very much modified and shortened, and I have no doubt her life prolonged four or five days. On one occasion Mr. Hetley, who watched the case with the most praiseworthy assiduity, allowed the convulsions to go on unchecked, and they then lasted a quarter of an hour; but when the chloroform was inhaled, they generally stopped in from two to four minutes. In neither of these cases, unhappily, could we obtain a post-mortem inspection, as is too often the case in private practice, unwisely and unfairly for medical science.

A similar hemiplegic convulsive state is met with after injuries to the brain. "We find it," says Sir Benjamin Brodie, "occur in cases of

of contraction showed itself in the muscles, and now they are not only attenuated but stiffened, as you may often see them in the dead body during the continuance of the rigor mortis. The attempt to extend the flexed joints is encountered by a powerful resistance, which can be only partially overcome, and always excites pain. In its lowest degree, the rigidity in this form of hemiplegia is limited to the flexors of the fingers. There are few cases, indeed, of long-standing paralysis which do not exhibit some degree of flexion of the fingers, resembling very much that caused by rigor mortis in the dead body. In the lower extremity the stiffness is most in the hamstring muscles, and in those of the calf, and the tibialis posticus and the flexors of the toes, and the biceps femoris. In its lowest degree it will affect the flexors of the toes or the hamstring muscles, or both.

As the process of contraction shows itself, in general, most in the upper extremity, so also it usually commences there; but now and then it will begin in the lower extremity: not unfrequently it will be met with in the upper extremity only.

The view, which I have always taken (and which many of you have often heard me express in passing through the wards) of the manner in which this contraction is produced, is as follows: at the seat of the original lesion, whether it be

simply a white softening, or an apoplectic clot, or a red softening, with more or less destruction of the brain-substance, there takes place an attempt at cicatrization, more or less perfect. Attendant on this, there is a gradual shrinking or contraction of the cerebral matter, which, acting on the neighbouring healthy tissue, keeps up a slow and lingering irritation, which is propagated to the muscles and excites in them a corresponding gradual contraction, while at the same time their nutrition becomes seriously impaired by the want of proper exercise, and the general depressing influence of the lesion.

Case XXXVI. Many years ago I watched the case of a young girl (A. M. Dawson), aged 20, who had been the subject of rheumatic fever and extensive endocarditis of the mitral valve. This girl became suddenly hemiplegic on the right side, with loss of consciousness and relaxed muscles. She recovered from the attack, and regained a very slight power over the leg. The muscles of the paralysed limbs wasted very much, and in the course of a little time they began to contract; the fingers were bent into the palm, and the forearm bent upon the arm; the leg, likewise, became rigid. Soon after this she began to have some epileptic fits at short intervals, the convulsions being confined to the right side. These fits were of the most fearful kind, consist-

punctured and wounded brain, where there is no pressure; and it so happens, when it has fallen under my observation in cases of depression of bone or extravasated blood, and where the exact nature of the injury has been afterwards ascertained, that the pressure has been always found to be complicated with wound or laceration of the substance of the brain."*

Convulsive movements of a less violent kind, more like the movements of chorea, occur in connexion with softening of one hemisphere of the brain. Thus, a man of fifty years of age, who had suffered from gout very much, became subject to fits of wandering; he would talk incoherently and seem lost, but after a little time recover; there were also chorea-like movements of the arm and leg of the right side. There were numerous spots of ossification in his radial arteries. After a free bleeding he became comatose, and sank. On examination, the grey and white matter of several convolutions of the left hemisphere were in the state of colourless softening, easily broken up by a stream of water. There were several osseous spots in the arteries of the brain, especially in the middle cerebral.

Not long ago, we had, in the Rose ward, a man named Fleming, æt. twenty-nine, in whom these choreic movements of the left arm, with

* *Med.-Chir. Trans.*, Vol. XIV. p. 352.

an imperfectly paralysed state of the upper and lower limbs on that side, accompanied a very weakened and deranged intellect. Any one who simply watched him as he lay in bed, his arm moving unceasingly, would have set the case down as one of chorea. To add to the resemblance, the symptoms originated in fright, caused by his house taking fire. Three days after the fire his illness began with these movements; he remained a long time in the hospital, becoming only more and more imbecile and deranged, and he was ultimately sent to St. Luke's.

In cases like these, as doubtless also in chorea, the perverted nutrition of the brain occasions irregular developments of the nervous force, which are tantamount to a state of irritation, and give rise to the convulsive twitchings. In chorea, the alteration is not so profound as to be beyond repair; in the softening of adults, unhappily, the lesion is too often beyond recovery.

There is one point with regard to this rigid condition of the paralysed muscles in this form of hemiplegia, to which I must refer before I conclude. It is this—that the rigidity is always, so far as my experience goes, much greater in the upper than in the lower extremities; indicating that the irritation is propagated from the brain to the spinal cord, and that the upper limb exhibits a greater degree of the muscular affection, because it is nearer the seat of irritation.

I have yet to consider the pathology of those cases of hemiplegia with rigidity of the paralysed muscles, in which that phenomenon comes on gradually, and some time after the paralytic seizure.

LECTURE XII.

On Hemiplegia with late Rigidity of the Paralysed Muscles.

In my last lecture I described to you the phenomena of hemiplegia with early rigidity of the paralysed muscles.

I showed you that in the first form of this kind of hemiplegia in which the rigidity is slight, and confined to the biceps of the arm, or at most to the biceps and triceps, and in a very trifling degree to the flexor muscles of the fingers, the paralysis is generally dependent on a clot which has lacerated the brain, and which is frequently preceded by white softening. The second form, in which the rigidity is considerable, and sometimes tetanic, is due to a more decidedly irritative lesion of the brain, or of its membranes, which is often inflammatory. The various cases which I cited showed, that although the lesions capable of producing such a hemiplegia differed, they agreed in one point—that they excited and maintained an irritated state of brain.

Now the prognosis in this form of hemiplegia is, on the whole, unfavourable: many of the cases die pretty soon after the attack, especially when

the brain-lesion is complicated with a sanguineous apoplectic effusion. As a general rule, the larger the clot the shorter will be the duration of life. Patients will survive an attack of this kind from a few hours to two or three weeks. *Perfect* recovery is, I suspect, extremely rare; indeed, I doubt that it ever occurs, excepting after inflammatory softening of very limited extent. In a few cases the state of rigidity gives place to that of relaxation, and the limbs remain relaxed and paralysed for the remainder of life—the muscles becoming wasted to the last degree. In one patient of mine (Cochrane, vol. xii.), in whom the paralysis and rigidity depended on inflammatory softening, the deltoid muscle and the scapular muscles of the shoulder-joint became so relaxed and wasted, that the head of the humerus dropped away from the glenoid cavity.

Many cases recover a slight amount of power in the paralysed limbs, after the shock of the palsy-stroke has passed off. But this improvement is not progressive, and, after the lapse of time, the muscles waste, the rigidity remains or increases, and the limb is permanently more or less flexed. In short, the paralysed limbs pass into the condition of what I would call *late rigidity*.

This leads me to bring before you the phenomena of hemiplegia with late rigidity of the paralysed muscles—to give you examples of it, and

offer some explanation of its pathology. It may follow the hemiplegia with relaxed muscles, as well as that with early rigid muscles.

I have already described a well-marked example of this form of hemiplegia in the case of Shea. (Case XXXI.) In this man, who frequently presents himself at the hospital, you have a favourable opportunity of observing the peculiar condition of the paralysed limbs. In the first place, you will perceive that the muscles are wasted; next, that the limb is in flexion, and even in extreme flexion; the arm is strongly adducted to the side, the forearm bent upon the arm, and the fingers bent into the palm of the hand. In extreme cases this state of the fingers is often attended with great inconvenience, from the irritation to the skin of the palm caused by the growth of the nails. Lastly, the muscles are tense like cords.

It is remarkable that in this, as in the palsy with early rigidity, the rigidity is most marked in the upper extremity, which is nearest the seat of the paralysing lesion.

In the history of Shea, you have that of nearly all the examples of this form of hemiplegia in its highest degree. He, you remember, fell in an apoplectic fit, and came out of it with hemiplegia of his right side, with all the muscles relaxed except the biceps. He regained power slightly, but after some time the process of wasting and

of contraction showed itself in the muscles, and now they are not only attenuated but stiffened, as you may often see them in the dead body during the continuance of the rigor mortis. The attempt to extend the flexed joints is encountered by a powerful resistance, which can be only partially overcome, and always excites pain. In its lowest degree, the rigidity in this form of hemiplegia is limited to the flexors of the fingers. There are few cases, indeed, of long-standing paralysis which do not exhibit some degree of flexion of the fingers, resembling very much that caused by rigor mortis in the dead body. In the lower extremity the stiffness is most in the hamstring muscles, and in those of the calf, and the tibialis posticus and the flexors of the toes, and the biceps femoris. In its lowest degree it will affect the flexors of the toes or the hamstring muscles, or both.

As the process of contraction shows itself, in general, most in the upper extremity, so also it usually commences there; but now and then it will begin in the lower extremity: not unfrequently it will be met with in the upper extremity only.

The view, which I have always taken (and which many of you have often heard me express in passing through the wards) of the manner in which this contraction is produced, is as follows: at the seat of the original lesion, whether it be

simply a white softening, or an apoplectic clot, or a red softening, with more or less destruction of the brain-substance, there takes place an attempt at cicatrization, more or less perfect. Attendant on this, there is a gradual shrinking or contraction of the cerebral matter, which, acting on the neighbouring healthy tissue, keeps up a slow and lingering irritation, which is propagated to the muscles and excites in them a corresponding gradual contraction, while at the same time their nutrition becomes seriously impaired by the want of proper exercise, and the general depressing influence of the lesion.

Case XXXVI. Many years ago I watched the case of a young girl (A. M. Dawson), aged 20, who had been the subject of rheumatic fever and extensive endocarditis of the mitral valve. This girl became suddenly hemiplegic on the right side, with loss of consciousness and relaxed muscles. She recovered from the attack, and regained a very slight power over the leg. The muscles of the paralysed limbs wasted very much, and in the course of a little time they began to contract; the fingers were bent into the palm, and the forearm bent upon the arm; the leg, likewise, became rigid. Soon after this she began to have some epileptic fits at short intervals, the convulsions being confined to the right side. These fits were of the most fearful kind, consist-

ing of very sharp clonic spasms, succeeding each other with extraordinary rapidity, and leaving the patient in an exhausted condition. After repeated attacks of this nature she died. On examination there was found a small cyst, containing serous fluid. This cyst was situate in the left hemisphere, just outside the optic thalamus. Its interior was lined by a yellowish matter, outside which, for some distance, the brain-substance was very much indurated. This was, no doubt, the contracted cyst and cicatrix of an apoplectic clot, the gradual shrinking of which operated as an irritant foreign body on the brain. Hæsit lateri lethalis arundo.

I shall give you a case similar to that of Shea, and also, no doubt, the same as to the nature of the lesion.

Case XXXVII. Mr. H., æt. 59, a healthy man, had been latterly working harder than usual, having unexpectedly become head of a large mercantile firm. On the 9th of June, 1852, he dined out, but as he felt unwell he ate and drank very sparingly; in the middle of the night he got out of bed to pass water, and suddenly became weak on the right side, and giddy; there was no loss of consciousness, and but slight impairment of speech. He was immediately attended by Mr. Robert Brown, of Brixton Hill, who cupped him on the back of the neck and purged him. After this he

quickly recovered; but at two o'clock P.M., on the 10th, he became completely hemiplegic on the right side, including the face and tongue, and lost entirely the power of speech, retaining that of deglutition, without any loss of consciousness. At six P.M., I saw him, and found him hemiplegic on the right side, with relaxed muscles, except slight resistance of the biceps; there were good reflex actions of the leg; he was quite speechless, and protruded the tongue with deviation to the right; the sphincter ani was partially paralysed.

It was evident that a very grave lesion had occurred in this case, sufficient to inflict so severe a shock on the brain as to destroy the power of speech, and to impair the power of the sphincter ani. Consciousness was retained, although the patient was lethargic. I think there must have been a good deal of white softening of the fibres connecting the convolutions and the corpus striatum; it is probable, too, that there were clots near the surface of the brain, but too small to produce such compression of the brain as would generate loss of consciousness.

On the 13th of June, at noon, just after passing water, this patient had a general convulsion, after which he remained for some hours in a lethargic, soporose condition. From this time he began to recover very slowly; he became more lively, and regained a very slight degree of power in the leg and arm; the face-palsy also diminished. On

the 16th of June he was still speechless, but understood all that was said to him, and made an attempt to write with his left hand. On the 4th of September, his general health was much improved; he was wheeled out in a chair every day, and remained very much in the open air. The paralysed limbs had become much wasted, and now we observed considerable shortening and stiffening of the biceps. On the 16th of March, 1853, intelligence was good; general health improved; and he made a fair attempt to sign his name. The paralytic limbs were much wasted; both *arm and leg had become semiflexed and rigid; the fingers were bent into the palm, and could not be perfectly extended.*

This case affords a striking instance of the progressive change in the paralysed limbs, proceeding probably *pari passû* with a cicatrizing or contracting process in the brain, and which is therefore slow and very gradual in its development.

Let me place in juxta-position with this case another very interesting one, in which I had the opportunity of watching the phenomena during life, and of ascertaining the condition of the brain after death.

Case XXXVIII. A lady, aged 60, was seized in the night of Oct. 6th, 1844, with hemiplegia of the right side, without muscular rigidity, with coma and stertorous breathing. She was quickly

attended by Mr. Dunn, who bled her and gave purgatives. In a few months she recovered completely, regaining the full use of the arm and leg, but retaining the single peculiarity of using one word for another, and of not applying appropriate names to the things she intended to signify; she never afterwards called even her own daughters by their right names.

On the 17th of May, 1847, she had a second attack. She was found in the morning lying insensible on the floor of her bedroom. She was again, and for the rest of her life, hemiplegic on the right side, completely as to motion, but some sensation remained; *there was no muscular rigidity whatever*. She was likewise speechless.

This patient recovered her general health slowly. And as she improved in this respect, the paralytic limbs wasted; the arm was kept in a semi-flexed position, the muscles slightly rigid; the fingers were bent inwards upon the palm, *and it was with some difficulty they could be straightened.*

In this state she remained until the 14th of April, 1850, when a third sudden seizure occurred, and for a short time *the paralysed arm and leg* shook violently, and the left side now became paralysed as well. From this attack she never rallied, and died on the fourth day.

The cause of the fatal attack and the left hemiplegia was found to be a recent clot, with softening of the right corpus striatum. And there was

likewise abundant evidence of old disease of the left side of the brain, upon which the original hemiplegia of the right side depended. Here were shrinking and wasting of the optic thalamus; this body had shrunk to less than half its natural size, its upper surface being greatly wasted. There was likewise considerable softening (colourless) of the white substance of the hemisphere, of the corpus callosum, part of the corpus striatum, and of the fornix. In the softened brain substance there were numerous small vessels in a state of fatty degeneration, and also abundance of compound cells.

In this case the contracted state of the muscles of the paralysed limbs on the right side was no doubt associated with the shrunken condition of the left optic thalamus; and it is not improbable that, had a similar state of the corpus striatum existed, there would have been still more rigidity of the muscles. The softening of the hemisphere sufficiently explained the loss of speech; but I think that a large portion of the softening on this side must have occurred shortly before death, as was indicated by the violent agitation of the paralysed limbs which occurred at the time of the last seizure.*

* I saw this patient several times with Mr. Dunn, who has published an excellent narrative of the case in *The Lancet* of October 26, and November 2, 1850. From that narrative my account has been condensed.

In further evidence of the connexion between a cicatrizing process in the brain, and this state of late contraction of the muscles, let me refer you to a case recorded by Andral, of which the leading points are these :—

Case XXXIX. A man, æt. 71, suddenly lost his consciousness on the 15th of May, 1820. This loss of consciousness lasted only a few hours, but on his recovery, he found himself paralysed on the whole of the left side. On the 28th of June he was admitted into *La Charité*. He was still paralysed on the left side; there was "complete *immobilité*" of the left upper and lower extremities, with very obtuse sensibility of those limbs. About the middle of August, it was observed that the paralysed limbs were not only much wasted, but contracted; the forearm was strongly flexed upon the arm, and the leg upon the thigh. Towards the end of October, the upper extremity, remaining paralysed, ceased to be contracted, but the lower extremity still remained contracted. He died on the 28th of November.

In this case the lesion consisted in a cavity large enough to hold an apple of moderate size. It was situated outside, above and behind the corpus striatum, midway between the two extremities of the hemisphere. Its walls were lined by a cellular, dense, and resisting membrane, in the substance of which numerous vessels ramified.

This membrane adhered closely to the substance of the brain, which surrounded the cavity. The cerebral substance, around, for an extent of four or five lines, was softened.

It is evident that a distinct process of cicatrization had been going on in this man's brain. Latterly this had been arrested, and a softening took place, which was, no doubt, the cause of the resumption of the state of relaxation by the extremity.

I find in Romberg's book a highly corroborative case.

Case XL. A woman, æt. 73, applied for paralysis of the right arm and leg. The muscles were at first flaccid; subsequently they contracted so forcibly, especially at the hand and forearm, that the fingers could only be opened by using considerable violence; nor did they remain extended, but instantly returned to their former position, like elastic springs. Owing to the long continuance of the disease, depressions had formed in the palm of the hand, which were covered with a mucous membrane, and secreted a thick fluid of an offensive odour. The contraction of the foot was less forcible. Death ensued four years and a half after the supervention of the hemiplegia, which had become associated with dementia; and six months before the fatal issue, paralysis of the left leg occurred, with contraction of the muscles.

The left arm retained its mobility to the last. The lesion connected with the paralysis of the right side, where the late rigidity had appeared, consisted of an old apoplectic cyst, of the size of a cherry, near the surface of the posterior lobe of the left hemisphere, surrounded by indurated brain and lined by a membrane, and in the same hemisphere, near to the corpus callosum, a brown spot of indurated medullary substance, extending into the left half of the corpus callosum to the septum lucidum. The left thalamus was flattened and atrophied, and half an inch smaller than the right one.*

I am far from asserting that the evidence which I have now brought forward is sufficient to *prove* that late rigidity is due to a cicatrizing process in the brain.† Many further observations are needed before this view can be considered finally confirmed. There is no doubt many more cases might have been adduced, had I been able to ascertain certainly the early condition of the paralytic limbs. Enough has been said to show you that the distinction between *early* and *late* rigidity is well based; and to direct your attention to the different circumstances under which each form occurs. Opportunities do not frequently offer to

* Romberg, *On Diseases of the Nervous System* (Sydenham Soc. Ed.), vol. ii. p. 424.

† Dr. Abercrombie's case CXXII. appears to have been one of this kind.

a hospital physician, of watching cases of paralysis of very long standing. Patients afflicted with palsy are admitted in the early period of the attack, and are discharged as soon as it becomes chronic; and then, in too many cases, the further progress of the case is lost sight of. In workhouses and other institutions, where the disabled poor are received, opportunities often occur, of completing the histories of chronic paralytic cases, and of determining the precise nature of the lesion. To these institutions many of you may hereafter be attached as medical officers; and I trust you will not neglect to investigate this point. It is only by the well-directed industry of many labourers that we can expect to bring so wild and rugged a field as that of cerebral pathology into a more productive cultivation.*

With reference to the actual condition of the muscles in the paralytic limbs with late rigidity, I think it must be admitted, as I have already intimated, that the rigid state of the muscles is due primarily to an irritated or excited condition of the nerves, and that on the cessation of that

* Observations pursued like those of Turck will, no doubt, throw great light on the changes which take place throughout the cerebro-spinal axis in connexion with chronic lesion of the brain. I have not yet seen more than a brief abstract of his book, entitled, *Uber secundäre Erkrankung einzelner Ruckenmark-stränge.* Vienna. 1851.

irritation, the muscles might resume their relaxed condition, or that a similar result would follow the severance of all connexion between the muscles and the seat of cerebral lesion by section of the nerves. It seems to me, however, that after a long continuance of this rigid and shortened state, the muscles would become permanently shortened, and would assume a condition similar to that into which those about ankylosed joints are apt to fall—a condition from which they would recover very slowly, or not at all.

I must now pass on to consider some other forms of hemiplegia, and this will afford abundant material for another lecture.

LECTURE XIII.

On Peripheral and on Hysterical Hemiplegia.

The next form of hemiplegia which I shall notice, is that which I have termed "peripheral hemiplegia." This name, which is not a correct designation when the whole of the pathology of the affection is taken into consideration, is, nevertheless, a convenient one, inasmuch as it indicates the most prominent feature of the malady, namely, the mode of access of the paralysis. The palsy is at first of the hemiplegic character, but after a time both sides of the body become involved, and it seems to travel from periphery to centre. At the onset of the disease, the patient complains of a feeling of numbness in the arm or leg affected, although, usually, touch remains perfect for some time, and he can distinguish two points, as such, at very slight intervals from each other. There is probably some altered condition of the nerves of sensation; and, perhaps, of the sentient nerves of the muscles, rather than of those of the skin. After a time the patient finds that he cannot perform small actions with the diseased arm; he cannot button his clothes, nor pick up a pin with the one hand, as well as

with the other; then he notices that he cannot write so well as formerly; he has not the same command over his pen which he previously possessed, and his handwriting becomes tremulous. A failure in the temperature of the extremity next manifests itself: it feels colder than the healthy limb, and sometimes the thermometer indicates that it really is colder. If the weather be cold, the affected limb will suffer more than the sound one.

For some time the patient continues to be able to grasp with one hand as well as with the other; the disorder at this period seeming to consist chiefly in the want of power to adjust the muscles for the finer movements, but not for the coarser actions; after a time more or less inability to accomplish the latter also comes on. After a longer or shorter period, the leg goes through a train of symptoms similar to that which the arm has previously undergone; the patient drags it after him in walking; his movements become stumbling; and he speaks slowly and hesitatingly, but yet his mental faculties do not seem to suffer much. This may go on for many months, or even years, for these cases are exceedingly chronic; the other side of the body, in some instances, begins to be affected almost simultaneously with, or very soon after the first: but in others it may be a considerable time before it suffers. Whenever it may begin, it passes through a train of symptoms

similar to that which I have described. The patient now begins to stoop; he finds he cannot hold himself erect; and in some instances his gait is apt to pass into that which is known as symptomatic of the disease termed "paralysis agitans," all the limbs shaking more or less, the trunk being bent forward, and, at every attempt at walking, there begins an irresistible tendency to assume a running gait. Sometimes the few closing months of life are disturbed by occasional slight attacks of epilepsy, and the memory and other mental powers participate in the general bodily decay.

In some persons this affection seems to limit itself to one leg only, or one arm—more frequently, I think, the latter; and in these cases, it is accompanied by a trembling or shaking movement, always aggravated by emotion of any kind, or by depressing influences, such as overwork, trouble, or anxiety.

In some cases the trembling movement can be controlled by a strong effort of the will; in others, such an exertion increases the trembling, as it does in chorea. In the case of a hard-working lawyer, in very extensive practice, this symptom affecting the arm existed for a period of five years, the patient having been all that time under my observation. It never extended to any other limb, and the patient died of the effects of cancer in the right kidney.

The peculiar affection of the hand which prevents a patient from using his pen, while he can perform any other action perfectly, seems allied to this. In one case of this disease I have seen both hands attacked. The patient had acquired the power of writing in an exquisitely beautiful style with the left hand, when it became affected in the same way as the right hand. In some cases the sentient nerves alone seem affected for a considerable period prior to the motor derangement.

Case XLI. I have here the notes of one of these cases, in which the early symptoms affected both sides equally, although one side was ultimately more weakened than the other. The patient was a man of the name of Barber, thirty-eight years of age (vol. xxvi., A); and I may here remark that so far as I have seen, this malady generally comes on at a more advanced period of life than in this case, the patients being almost always above the age of fifty. This man had always enjoyed good health; he had lived well, and had never had syphilis. The disease commenced six months before admission, with a sensation of tingling and pricking in both hands (what is commonly described as "*pins and needles*"), with a considerable amount of numbness about both elbows, just as if the ulnar nerve had been jarred, or, in the patient's own words, "as if he had struck his funny-bone." With all

this, there was a sensation of heat in the forearms; and he soon became unable to button his clothes, or pick up any small object with the right hand. Under treatment, the tingling and numbness became diminished, but the loss of power continued, affecting the right side chiefly; this increased to such an extent, that he could not raise the right arm above the shoulder. When he came to the hospital, he could scarcely grasp with the right hand, and that with difficulty; he could only raise the arm to the level of the shoulder, and dragged the right leg considerably in walking. There was some deficiency of power in the left hand also, but not so much as in the right, and he could raise that arm perfectly; the power of the left leg was not impaired. The morbid sensations which I have described existed on both sides. There were no pains in the head, nor giddiness; but he experienced a certain feeling of insecurity in walking.

He remained in the hospital some time, and was subjected to various plans of treatment, among which were free purging, and mercurializing; but without any other benefit than that he could walk more securely than on his admission.

Case XLII. I shall refer you to a second example of this affection, which occurred to me in private practice. The subject of it was a medical man, æt. 52, who consulted me early in 1849. He had noticed for some months before, gradually

coming on, a sense of numbness in the right arm —a sort of feeling as if it were not in its right state, while the faculty of touch was not impaired. Then he found himself unable to execute the finer movements, his handwriting became very shaky, and he was often compelled to employ an amanuensis. Hurry, excitement, or emotion always increased his difficulty, as regards these actions. After this, being engaged in the practice of midwifery, he found he could not manipulate with his right hand with the same facility as formerly; and this was, of course, a source of great inconvenience to him. All this time he could grasp very well, and with considerable power. Soon afterwards, the right leg became similarly affected, and it was then that I first saw him, now upwards of four years ago. There was no symptom immediately referable to the head; no pain, nor giddiness; memory good. This spring (1853), happening to be in that part of the country in which he lives, I visited him, and found that the disease had spread to the left arm and side, and that a train of symptoms had occurred on this side, precisely similar to that which had previously taken place on the right. His articulation was affected, having become thick, and rapid in the mode of utterance. His body was now beginning to be bent forward, and he had found it necessary to retire from practice; but his mental faculties were so far quite unimpaired.

Case XLIII. A very similar case occurred to me also in the previous year (1848), in the person of a country gentleman, æt. 64. He came to me one day from his club, complaining that he had, for some time, experienced increasing difficulty in buttoning his waistcoat, or his trousers, and consequently he used sometimes to leave parts of the latter garment open, in a not very seemly way. He complained of pain, and a numb sensation in the right arm, but for a considerable time his sense of touch remained unimpaired, and he was able to grasp with considerable power. After some time the leg became similarly affected; when I last saw him the disease had spread to the opposite side of the body, and he is at present almost bent double, and has for some time been obliged to give up writing. A great variety of remedies was tried both in this and the preceding cases; among them galvanism, without any effect.

The pathology of this affection is exceedingly obscure. I have never had an opportunity of making a *post-mortem* examination, as the disease generally goes on, for many years, without proving fatal to life. Both the patients, whose cases I have just related, are still living; but the man, who was under treatment in the hospital, we lost sight of, soon after his discharge. Nor do I know of any published account of a *post-mortem* examination of a case of exactly this kind, and I am therefore unable to offer any satisfactory hypothesis, as to

the pathology of this affection. I am inclined to think that it consists in some degree of atrophy of the nerves of the extremities, with a similar condition of some portion of the brain, either of which may stand to the other, as cause to effect. But from the mode of access of the disease, from the first symptoms showing themselves in the extremities, without the occurrence of any head-symptoms, I think we may, for the present at least, conveniently mark this disease *clinically* by designating it *peripheral hemiplegia*.*

* The late Dr. Cheyne describes, under the name of Creeping Palsy, a disease which I believe to be the same as this. He also gives the dissection of one case. I quote the whole of his description.

"First, there is observed numbness in the course of a nerve, often in the sciatic or in the ulnar nerve. This may exist for some months without any other symptom of disease, and, indeed, I believe the disease sometimes goes no further; but frequently, after some months, a slight defect—a drag, as it is called—is observable in one of the legs, which renders the patient liable to trip; next an inability to use one of the hands in such ways as require combinations of the muscles of a more complicated nature; for example, the patient cannot guide his hand into his coat-pocket. The pulse will be found slow, the circulation languid, the expression inanimate, together with restlessness. Some defect of mental power is discoverable; the apprehension is tardy, the speech less articulate, the words inappropriate, and the recollection of recent events not distinct. Then the disease proceeds more rapidly, the sphincters begin to fail, slight convulsions occur, the individual becomes hemiplegic, or complete paraplegia takes

Hysterical Hemiplegia.—Let us now inquire into the nature of the hysterical hemiplegia, or that form of hemiplegia which occurs in hysterical patients, without any apparent lesion in the brain. Hysterical paralysis is by no means of uncommon occurrence, the variety most frequently met with being that which is called "hysterical aphonia," wrongly termed *aphonia*, because the subjects of it are almost always able to speak in a whisper. This may be considered the type of this kind of paralysis. It frequently comes on very suddenly, and goes off just as suddenly as it came. A young lady, for instance, will go to bed quite well, but, on rising the next morning, she is unable to speak in her natural tone of voice. After a time her voice comes back suddenly, and it often returns under the influence of strong emotion. The loss of voice may go on for many weeks; indeed,

place, with imbecility of mind, the convulsions become stronger and more frequent, and in one of these the scene closes."

Dr. Cheyne gives one dissection of a case in which the paralysis was on the left side. "The medullary substance of both hemispheres of the cerebrum was melted down into a soft mass of the consistence of thick cream. The cortical part of the brain surrounding this substance was firm, and seemed condensed, so that the softened medullary mass seemed confined in a kind of cyst. The corpus striatum of the right side was softened like the medullary matter of the hemispheres; that of the left side was natural."—*Dublin Hospital Reports*, vol. iv. p. 269.

in the last case of the kind that I have seen, it continued upwards of three months. In some of these cases the patients are in a very weak state of health, but in others there is apparently no great deviation in this respect from the normal condition.

Let me mention to you here an instance of the sudden manner in which the lost voice will return in cases of this kind. The patient was a young lady who had lost her voice. She came to see me at my house several times; after having thoroughly satisfied myself as to the nature of the affection, I assured her that her voice would come back quite as suddenly as it went away,—some day, perhaps, when she least expected it. One morning she came to see me, as usual, and, having been reminded of what I had so often told her, she left the house; she had hardly walked ten yards from the street door, when she recovered her voice. Her sister wrote to me immediately, to acquaint me with this happy fulfilment of my prediction, and added, that on returning home her relatives would not believe but that I had been electrifying her, or performing some conjuring upon her; and, no doubt, had I shortly before tried some new remedy, or made some mysterious passes before her, one or other of these expedients would have had the credit of the cure. What you have to do, in such cases, is to satisfy yourselves, that there is no morbid condition of

the laryngeal mucous membrane, whether tubercular or otherwise, and if no seriously disturbed state of the system should arise, you may prognosticate, with certainty, that the voice will in due time come back. The subject of "hysterical aphonia" I have brought under your notice as exemplifying the typical form of *hysterical paralysis*, which, in these particular cases, consists in a weakened state of the nerves and muscles of the larynx. But *hysterical paralysis* sometimes affects one limb only, sometimes both lower extremities, constituting "*hysterical paraplegia*;" and sometimes, though certainly least commonly, the upper and lower extremities on one side, and then it will constitute "*hysterical hemiplegia*," a condition which, although its existence is denied by some authorities, still is sometimes, though rarely, met with. Indeed, if a patient lose the power over one arm, there is no reason why she may not lose that over the leg of the same side, and then you at once have a hemiplegic case.

Hysterical hemiplegia occurs in the same class of persons, under similar circumstances as other forms of hysterical paralysis. The period which immediately follows that of puberty, and that which precedes the change of life, seem to me most liable to hysterical affections. They are brought on by exhausting causes, such as excessive menstruation, leucorrhœa, over-work, anxiety or excitement, or indeed by any debilitating influences.

In the hysterical hemiplegia, neither the face nor the tongue is affected; the palsy is limited to the upper and lower extremity, and is often not complete; the muscles are generally relaxed, but do not suffer much in their nutrition, as compared with those of the other side; now and then one or both limbs may be affected with spasm of some of the muscles, or may have a tendency to pass into cataleptic rigidity. In walking, when the palsy is pretty complete, the leg is drawn along, as if lifeless, sweeping the ground.

Let me direct your attention to some examples of this affection. One occurred in the hospital some years ago, and I then gave a clinical lecture upon it, to which I must refer you for the details of the case, and my remarks on it. The patient's name was Leigh; she was at the later of the two periods of life which I have mentioned. (Vide Lect. i. p. 17.) There were in this case the peculiar mode of progression as regards the lower limb—the sweeping movement, so to express it—and the absence of face and tongue palsy, which, I think, characterise the hysterical affection. There were, likewise, no reflex actions.

Case XLIV. A second example of this affection was in the case of E. Somers (vol. xii., p. 66), at the early age of 21. This patient was brought into a state of great debility by typhus fever. The catamenia were very defective; she had not been unwell more than twice in the last four years.

After an accidental fall, in which she struck her head violently, the paralytic state first showed itself. The fall happened six months before her admission, and the paralysis occurred a month afterwards. The left arm and leg were affected, from which she recovered nearly completely, when another fall aggravated the evil, and she came into the hospital.

The peculiar sweeping movement in the leg was less marked in this case than usual, because there was a spasmodic state of the muscles which rotate the thigh inwards, and in consequence the patient kept her foot in an almost constant state of inversion. She nevertheless dragged it along as if it were lifeless, without raising it from the ground, or resting upon it in the least; nor was she able to walk without the aid of another.

The hysterical nature of this case was indicated by the frequent occurrence of paroxysms of hysteria, in which the patient would pass quickly into a state of nearly complete coma. In these attacks the paralysed limbs would be affected with spasms. It was certain that they were not epileptic, for they were not attended with the complete loss of consciousness which is characteristic of epilepsy. There was no face or tongue palsy; but while she was in the hospital the right side was, for a day or two, affected like the left.

This patient was subjected to a tonic treatment. She was made to walk at regular periods

daily, with the help of another, along the wards and corridors, and regained very much the power of the leg, the arm having very soon recovered itself. The hysteric paroxysms ceased, and she left the hospital in three weeks improved, having refused to submit to the application of galvanism to the leg.

Case XLV. Let me quote another example of hysteric palsy, to illustrate how the hemiplegic state must be regarded as a simultaneous loss of power in the upper and lower limbs on one side, rather than as a crossed influence acting from the brain. This case affords an instance of the palsy affecting one leg. The patient, Sarah Best, æt. 27, was married between three and four years before her admission (August, 1843). Since her marriage the catamenia had become very irregular and painful, and her health, previously good, had suffered very much. She became subject to flying pains in various parts of her body, and lost strength and flesh. She suffered, likewise, from frequent paroxysms of hysteria, and passed large quantities of pale, limpid urine. A month before admission she began to complain of pain across the upper part of her abdomen, which the least pressure greatly increased. It was treated by leeches, purgatives, and fomentations, and subsided. A week before she entered the hospital she experienced a dull, heavy pain in the left leg,

which impeded her walking. Next day the pain increased, and with it came a sense of numbness in the leg; the power of motion decreased very rapidly, and she was unable to use the leg in walking, dragging it along as if it were dead, being unable to move it in any other way. The catamenia had ceased for six months. There was no indication of brain or spinal disease, nor was there any palsy of any other part of the body. The treatment was directed to the restoration of the catamenia, and she was made to exercise the weakened limb. In a week she was able to walk about the ward, and was well enough to return home in a little more than a fortnight.

Another case similar to the preceding was that of E. Goldsworthy.

Case XLVI. This young woman was aged 28, admitted March 6th, 1850. She had lately come off a journey from India, where she had had the cholera; she was a person of distinctly hysterical temperament, and suffered much from leucorrhœa. For two years she had suffered from pain in the left hip, and during all that time she was unable to use the left leg as freely as the right. Shortly before admission the left leg became weak, and she had occasional numbness and twitchings in it; the leg, on her admission, was so weak, that she could not walk without help. In progression the leg was drawn lifelessly after her, sweeping the

floor; sensation was not impaired. The arm experienced the same kind of numbness and weakness as the leg, but to a much less degree. There was no face palsy.

This woman remained in the hospital upwards of three months, under a treatment by tonics, shower-baths, and galvanism. She left it much improved, but not quite well.

As it is important to establish the fact of the occurrence of such cases as this, let me add another example.

Case XLVII. M. Holdup, æt. 35. This woman has been a cook. Two of her sisters are nervous, the elder being affected similarly to herself; admitted March 19th, 1850. Three years before her admission she had a similar affection, and was cured by a change of air.

Three weeks before she came in, on returning home one evening, she suddenly felt great feebleness in walking, but contrived to get home and had to be carried up stairs; she was purged, leeched freely, and blistered behind the ears, but without benefit.

On admission the left arm and left leg were found very weak, the arm less so than the leg. She could move the arm, but with diminished power and some tremulousness of motion; the grasping power was also enfeebled. The leg was moved with feebleness and difficulty, and she

swept it after her in walking; her gait was very unsteady; the muscles of both limbs flaccid. There was in this woman a decidedly hysterical constitution, and the catamenia were scanty and irregular.

This patient benefited very much and quickly, by tonic treatment, shower-baths, &c., and at the end of ten days her power of walking was much improved, and she dragged the left leg much less. She was obliged to leave the hospital at this time.

Case XLVIII. Early in the present year (1853) I was consulted in the case of a lady about thirty-five years of age, who had well-marked paralysis of the hysterical kind. In this case there was excessive leucorrhœa and amenorrhœa, with occasional attacks of hysteria. In September, 1851, she caught a severe cold. Some days after this, on getting out of bed, she fell forwards on the floor, having lost the use of both her legs. She recovered the use of the left leg, but the right continued paralysed up to the time I saw her. At first there was a total inability to stand; she gradually recovered from this, and was able to use the leg in walking, but with the peculiar sweeping movement. It was a curious feature of this case, that at one time the *left* arm was paralysed, and the power of the hand was so affected, that she could not hold small articles, such as a pin.

Under a very careful treatment by Mr. Morley, of Barton-on-Humber, the uterine function was restored and the leucorrhœa reduced, and although a long time elapsed, she had regained all power but that of the leg, which still, in May, 1853, remained paralysed. Galvanism had been only partially used, and I recommended its continuance in a more effective form. This lady, I am informed, has been progressing favourably, under a course of muscular exercises, directed by Mr. Amesbury.

I have thought it important to bring before you this form of one-sided or hemiplegic paralysis, because it may so far simulate that from cerebral lesion, as to lead to serious mistakes in practice. A very serious mistake would be to pronounce the paralysis from brain lesion to be hysterical; and this is the more possible, as a state of hysteria may co-exist with brain lesion, which may give to the paralysed limbs much of the hysterical apparent lifelessness.

The two following cases of hemiplegia are examples of its occurrence in young women at an age when hysteria is most developed, and most apt to simulate organic disease. One recovered, the other died. Both resembled the hysterical affection in many points, and for this reason I trouble you with the details of them.

Case XLIX. Caroline Willis, æt. 25 (vol. xviii.

p. 25), employed in bookbinding; always a resident of London; unmarried. The following is the abstract of her case from the case-book; date, June 2nd, 1846:—For the last three or four months she has had general weakness, headache, and drowsiness, for which, one month ago, she was bled to one pint. The weakness increased, and two weeks afterwards she had an hysterical fit during the night. She jumped out of bed, threw her arms about, and laughed very much. This lasted about half-an-hour, when she became perfectly rational. After this her mother noticed some impediment in her speech, which increased up to May 29th, when she quite lost her speech. Ten days before admission her right arm and leg became quite paralysed, with relaxation of the muscles, and without loss of consciousness. The arm was completely paralysed; the leg not so much so; she could use it in walking. The right side of the face was paralysed, and the tongue deviated to the right; speech much affected and deglutition impaired; manner childish. It was ascertained that she was affected with gonorrhœa at the time of her admission. She complained of pain in the head, chiefly in the forehead. The right pupil was larger than the left.

In consequence of her great weakness, when admitted, she was put on quinine, after a slight purgation. In seven days she had improved considerably. She could walk better and swallow

more easily. As it was feared there might have been some syphilitic taint, she was ordered calomel and opium. Under this treatment she continued to improve; she soon gained the complete use of the leg; that of the arm came much more slowly. This mercurial treatment was continued for a week only, and she then took ammonia and bitter infusion.

She remained in the hospital till the end of July, and was then discharged very much better in all respects.

In this case there was evidently either cerebral or meningeal lesion, which it is not improbable may have had its origin in syphilis.

Case L. The second case is of recent occurrence, and excited great interest from the early age of the patient, and from the many points of resemblance to the hysterical affection which we observed in it. Some of you will recollect how closely we watched the case, hoping to find some sign which would unequivocally indicate its real nature, and especially as to the more favourable view of the case. The patient, Mary Ann Hopkins (vol. xxxix., p. 130), was nineteen years of age; she came into the hospital the 28th January, with incomplete paralysis of the right arm and leg, also of the right side of the face—the muscles being flaccid. The tongue did not exhibit any signs of paralysis. The pupils were equal.

Her mode of progression resembled that which I have described in the hysterical palsy, as she seemed to sweep the foot along the floor as she walked. The power of the arm and hand was not so much weakened as to prevent her doing many useful things. Thus, on the 29th of January it was reported: "She can dress herself, and fasten her gown without any difficulty, and she was observed yesterday arranging her back hair, but she complains that she cannot do any neat work, her fingers feel numbed, and she can scarcely feel the needle which she holds. She complains of numbness and weakness of the right side, and a sense of weight in the arm. The grasping power is much impaired, and the sense of touch is less acute than on the left side; she has no headache, and cannot assign any cause for her illness; no evidence of heart disease; her appetite is very good, almost ravenous. Bowels regular.

Now the history of this girl was as follows:— She was of healthy parentage; was born and lived all her life in a village in Kent, not far from London. Her general health had never been very good. Three years before her admission she had had typhus fever. She was naturally of a nervous disposition, subject to great variation in her flow of spirits, and suffered from frequent attacks of giddiness. She had menstruated regularly for the last three years, at intervals

of three weeks, and lately the discharge had been very profuse, continuing for about four or five days. There was no leucorrhœa.

Two months before her admission she suddenly felt her right foot, while walking, become numb and weak, and she began to halt a little on that side. The palsy and numbness gradually extended up the whole of the leg and thigh. The right arm now became similarly affected, and felt cold and heavy; the features became at the same time slightly distorted, and the speech a little affected.

You will remember that in discussing the point, as we went round the wards, as to whether the hemiplegia was, or was not, of the hysterical kind, I stated that the existence of facial paralysis went very far against the diagnosis that it was of that character. For a time I was much tempted to take the latter, more hopeful, view of the case, and to this I was led by the resemblance of the mode of progression to that which I had already previously noticed in hysterical cases; and also by the early age of the patient, a period when so extensive hemiplegia does not often exist, and by her catamenia being irregular; also by her having latterly become subject to hysterical fits of a very severe kind, and her sisters having been likewise subject to similar attacks. Another feature noticed in this case, and also in hysterical palsy, was the inability to excite reflex actions, but no reliance could be placed on this as a diagnostic sign. After

a time, symptoms occurred, which rather militated against this diagnosis; the patient became subject to attacks of violent sickness, which would last for two or three days, and were so severe that, during this time, everything was rejected by the stomach. I may remark here that nothing seemed to relieve these attacks of sickness so much as free purgation by large doses of calomel. At the same time the hysterical paroxysms increased in frequency and severity, the attacks were so severe as to border on the epileptic, but we tested her sensibility on several occasions and found that she always evinced sufficient consciousness to show that they were not of the latter kind. I will read to you the description of one of these attacks, by Mr. Plowman, my clinical clerk. It occurred on the 8th of February. "At the visit she was found in a very severe and continuous fit, in which she had been, the nurse said, for upwards of an hour; she had passed her water under her, and was rolling about, and appeared suffering violent contortions of the left side of her body; but she was evidently conscious, as when the bed-clothes were turned down, she repeatedly endeavoured to replace them, though with uncertain jactatory movements; and when her legs were pinched, her countenance gave marked indication of feeling, which was equally expressive, on whatever side she was tried."

After she had been a fortnight in the hospital, the paralysis increased, and at length became com-

plete, the relaxed condition still remaining. At length she died in one of the hysterical paroxysms, eight months after the attack of paralysis. On examination of the brain after death, the left hemisphere was found to be the seat of considerable lesion, which appeared to be due to the deposition of tubercular matter about midway between the convolutions on the outside and the corpus striatum and optic thalamus within. There was a large amount of softening of the brain-substance, all around the deposit, so that the white matter seemed diffluent, and fell away, leaving a large cavity, just outside the lateral ventricle. The disease, no doubt, consisted in the gradual deposit of this scrofulous matter. So long as this was small, and the brain-substance around it healthy, the palsy was slight; but as soon as the cerebral fibres, especially those going to the corpora striata, became softened, and altered in their nutrition, (under the influence of the pressure of the scrofulous matter,) the paralysis became complete.

In this case we had an example of the hysterical diathesis highly developed, and complicating the effects of cerebral lesion. The difficulty of the diagnosis was at one time much enhanced by the exquisite character of the hysterical phenomena, and to a degree which cannot be fully appreciated by those who have only heard or read the details of the case, and did not see the patient. The result confirmed the value of face-palsy, as a dis-

tinguishing symptom of the paralysis of brain-lesion from that of hysteria.

The diagnosis of *hysterical paralysis,* whether it be of the hemiplegic or paraplegic form or whether one limb only be affected, depends on these points :—

1st. The hysterical constitution of the patient herself and of her family ; and there are certain signs which, as you know, are held to be indicative of the *hysterical diathesis,* such as a lax condition of the tissues generally, a peculiar fulness of the upper lip, drooping of the upper eyelids, &c.

2nd. The absence of signs of lesion of the nervous centres.

3rd. The characters of the paralysis itself; the absence of palsy of the face and tongue ; the peculiar movement of the leg in progression ; the fact of the paralysis not being complete, the muscles not being so much wasted, and the fact of the patient being sometimes able, under the influence of strong emotion, to use the paralysed limb as well as the sound one, or nearly so. But you will not fail to recollect that, even in decided lesion, the paralysed arm is sometimes moved in yawning or sighing, or under strong emotion.

In concluding this already too long lecture, I shall refer briefly to a kind of hemiplegia, of which I have seen but little, but which is sufficiently marked to demand special notice. It occurs in

men of hypochondriacal habits, and in women too. In its mildest form, it may be distinguished as "emotional paralysis," or when it affects one side of the body, "emotional hemiplegia." It most commonly consists in a simple loss of speech, occurring under some strong excitement, the power of speaking returning usually in a few days, and, indeed, generally very rapidly after the patient has regained the ability to pronounce one or two words, such as "yes," and "no."

The following case affords a good example of this affection in both forms.

Case LI. The patient was a man between fifty and sixty years of age, of irritable temper and hypochondriacal habit. A question, respecting some very trifling matter, happening to arise one evening in his family-party, some one present held out too strongly against his view, and this led to a vehement contradiction on his part, which was met by a counter-statement and a rejoinder, and thus he became excited to such a degree, that his power of speech completely abandoned him.

Every one knows how mental emotion and excitement tend to choke the power of utterance; you will remember how well Virgil makes Æneas express the combined influence of grief and terror in the line—

"Obstupui; steteruntque comæ; et *vox faucibus hæsit.*"

Men of irritable and excitable temper, under

the influence of strong passion, are apt to stammer and stutter, and find it difficult to give expression to what they mean. It is just this, that occurs in the cases to which I am referring, only in a much greater degree. But the fact, that recovery takes place speedily, indicates that no considerable amount of lesion can have occurred. Whatever, indeed, that may have been, it must be looked at as a lesion, caused by powerful mental influence, and partaking very much of the nature of *shock* affecting some part of the brain.

This view of the case was corroborated by other features of it. The patient had full use of his muscles; he had full power over his hands and feet; he could sign a check, and his mental faculties seemed unaffected; only he could not speak, and whenever he tried to do so, the attempt would end in a fit of crying. He continued in this speechless state for about a week, when he recovered, and when once he began, the power of speech returned fully in a very short time. Two years after this occurrence, the same gentleman got into a similar argument, and difference of opinion, upon a matter equally trivial, and became again strongly excited; but this time, instead of becoming speechless, he became hemiplegic on the left side, without mental affection, but with decided palsy of the left side of the face. The paralysis was not complete, for he could move the fingers and leg very slightly. After a little time, without any other treatment

than that of removing as far as possible all exciting causes, he recovered to a great extent the power over the arm and leg; but, although the principal recovery took place about six weeks after the attack, he is now, four months after the occurrence of the hemiplegia, by no means quite well. To what extent lesion has taken place in the brain, in this case, I cannot take upon myself to say, having never had an opportunity of examining, after death, the brain of a person who had suffered from this affection; but I am disposed to think that, if there be any lesion, it must be slight and, probably, allied, in its nature, to that which occurs in those transient attacks of hemiplegia, which occur after epileptic seizures. Of these I shall speak in my next lecture.

LECTURE XIV.

On Epileptic Hemiplegia.

There are yet other forms of hemiplegia, which clinical observation will teach you to recognise, and which, for the sake of prognosis and treatment, it is highly important you should clearly distinguish from those which I have described in the preceding lectures.

The most common of these is that which follows, and is caused by the epileptic paroxysm, and which, to mark its relation to the fit, I would designate "*Epileptic Hemiplegia.*"

The history of the more simple cases of this kind is just this: A patient has a fit, distinctly of the epileptic kind; he comes out of it paralysed in one half of the body; generally that side is paralysed which has been more convulsed than the other, or which has been alone convulsed; but the paralysis may occur where both sides have been convulsed equally. The paralytic state remains for a longer or shorter time, varying perhaps from a few minutes or a few hours to three or four days, or even much longer. It then goes off, or improves, until the next epileptic fit, when a train

of phenomena, precisely similar, recurs with like result.

Let me give you some illustrations of this epileptic hemiplegia.

Case LII. A good example of this occurred in Sutherland ward in February last (1853). The following are the chief points of the case: Jonathan Woolley, æt. 10 (vol. xxxviii., p. 188), had convulsions when teething; since that time he continued healthy, till the end of 1852, four months before admission. He then had his first fit, which came on without apparent cause: he seemed equally convulsed on both sides of his body. In all the subsequent fits the convulsion has been confined to the right side, and after each, the patient was distinctly paralysed on that side, with relaxed muscles. The paralysis was of motion only, and was not complete, a slight amount of power remaining. This boy's fits were of short duration, not lasting above four or five minutes, and the remaining coma was also very short. The intervals between the fits varied very much; sometimes he would have several at once, as many as eight in a day, and this was more likely to occur after a long interval of freedom. The boy remained three weeks under treatment by good food, cold water splashing, and occasional aperients, and he continued free from fits during the last fortnight of his stay in the hospital.

Case LIII. Thomas Orton, æt. 34 (vol. xxx., p. 80). This man had been healthy up to fifteen years before his admission in March, 1850. In the year 1835 he was thrown out of a chair, and pitched violently on his head. He was stunned, and his left arm contused, but recovered perfectly. A year after this he was suddenly seized with a sensation as of an electric shock traversing the whole of his left side, and he fell down insensible, foamed at the mouth, bit his tongue, and was convulsed. From this he speedily recovered, but with *his left arm and leg paralysed;* the face was paralysed, and the tongue greatly lacerated, on the left side. These fits recurred daily for about ten minutes, and were always ushered in by twitchings in the fingers of the *left* hand. The paralysis had never perfectly recovered since the first attack; but after each fit it became worse for a few days, and then recovered to the point at which it existed before the fit. There were also in this case minor fits, the patient experiencing a sudden start and falling down for an instant, with a vacant look, but not insensible. The weakness in the left side is not at all increased by these smaller attacks. This patient had been treated by nitrate of silver, and his skin was discoloured in consequence. He remained in the hospital two months without any material change in his symptoms, excepting that the intervals between the attacks seemed to be prolonged. He was treated

with the tincture of sumbul, and afterwards with valerianate of zinc.

Case LIV. H. Pitt, æt. 26, a labourer (vol. xxxiv., p. 84), had fits at irregular intervals for eight years, from which he recovered very quickly, so as to be enabled to resume his work on the same day. In each fit he became paralysed on the right side with relaxed muscles. The face was paralysed on that side, and his speech was slightly impaired. The paralysis was never complete. It did not improve for eighteen months previous to his admission, December 9th, 1851, but became worse after each fit, and recovered to a certain point before the next. It was ascertained that the fits were the effect of intemperate habits; and evidence was obtained of a syphilitic taint. He was subjected to a mild course of blue pill for twenty-eight days, and left the hospital very much recovered.

Case LV. Mary A. Godfrey, æt. 29 (vol. xxxi., B, p. 147). No positive evidence of syphilitic taint could be obtained, although upon that point I had strong suspicions. Her first epileptic fit was in January, 1850; the second early in March. She came out of this fit with paralysis of the left arm and leg and side of the face. This attack of paralysis lasted only half an hour. In three weeks afterwards she had another fit with like results. In the night of the 16th of May she had a fit in

her sleep, and awoke up in the morning with the left side paralysed, including the face and tongue. The paralysed parts had their sentient power very much diminished, and there was ptosis of the left upper lid. She was admitted into the hospital on the 21st of May. Under the use of quinine, iron, and iodide of potassium, and occasional purgatives, she quickly regained the power of the paralysed limbs, both as to sensation and motion, and left the hospital quite well in seven weeks, notwithstanding an attack of pericarditis, which came on after she had been three weeks in the house.*

The next case affords an instance of paralysis of one arm, consequent upon the epileptic fit.

Case LVI. Ellen Biddlecomb, æt. 24 (vol. xxx., p. 130), admitted April 3rd, 1850. Catamenia disappeared three years ago, and soon after she had her first fit. She suddenly fell, and was convulsed and insensible for half an hour. She came out of the fit paralysed as to her left arm, but recovered it the next day. The fits, since this first one, have recurred generally twice a week, although there has been as long an intermission as three weeks. After every fit she was found paralysed in the left arm, and she suffered excessive pain in it. Both the paralysis and the pain always subsided in one or two days.

On the 6th of April, three days after her ad-

* Vide p. 187.

mission, she had a fit, and the following note was made: "Last night she had a fit, which appears to have been less severe than any previous one, having lasted only for ten minutes. This morning she can scarcely move her left arm, which is likewise very painful." Similar reports were made on May 11 and June 11. The arm recovered in two days, and the pain was relieved by chloroform applied locally. This girl was of a decided strumous diathesis, and had enlarged cervical glands; she derived benefit from the treatment in the hospital. She had shower-baths, cod-liver oil, and sumbul. The fits were suspended for a whole month.

Case LVII. Some of you will remember the case of an old soldier, Arnold Young, æt. 58 (vol. xxxv., p. 83), who was admitted on the 29th of October, 1851. This man's first fit was in February, 1851; of this we have only a very imperfect account. Of the subsequent fits, each was ushered in by a numbness and twitching of the right great toe, extending gradually up the leg and side to the arm, which was also affected. Both leg and arm would then be seized with convulsive twitchings. Very often the attack would end there: and he stated, that, sometimes, by having the arm and leg diligently rubbed, or, less effectually, by tying a tight ligature around the arm and thigh, the fit of general convulsion would

be prevented from coming on; and, in fact, this happened several times while he was in the hospital: when he felt the warning he would throw himself on the bed, and rub the arm and leg, and employ others to do so, or apply the ligatures, and frequently with success. If, however, these preventive means failed, the fit would come on, with convulsions and coma, the former affecting chiefly the right side, and leaving the right arm and leg paralysed, and also the right side of the face and tongue. He had not had a fit for between four and five weeks prior to his admission into the hospital; the right arm and leg, which were rendered utterly powerless by the last fit, had, on his admission, regained a considerable amount of power. He could raise his hand to his head, but the power of grasping was very feeble, and he could not use the hand to cut his meat; he walked with a limp. The recovery of the leg was more rapid and complete than of the arm.

From this man's admission to the 30th of November he had three times a threatening of a fit, which was stopped each time by friction. On the 30th he had a complete attack, notwithstanding the application of ligatures. He was very much convulsed; and, on recovering, the right side, which, in the absence of the attacks, had regained power very much, was greatly weakened. From this time till the 12th of January, he had two threatenings, and one fit.

The treatment consisted in free counter-irritation of the scalp, on the left side, by tartar emetic ointment, and the use of iodide of potassium. Further than that he gained power in the paralysed limbs, he did not seem to derive marked benefit.

Case LVIII. A very good example of this form of hemiplegia is afforded by the following case:—Sarah Bone, æt. 63, admitted September 17th, 1853. I shall quote the notes of the case made by Mr. Liveing: "She is a charwoman, of tolerably healthy appearance for her age, thin, and rather pale. She has always enjoyed good health, excepting that she had been subject to severe headaches, as she says, 'seldom getting up in the morning, or going to bed, without one, since her childhood,' until she ceased menstruating, since which time they have disappeared. The headaches used to be always most severe on the right side of the head.

"Last November (1852), after having been heavy and sleepy for some days, she had a fit; her daughter states that she foamed at the mouth slightly, and that her left hand was convulsively clenched. From this fit she recovered with paralysis of the left side of her body; but in a short time she was able to walk about again, only experiencing a numbness and sensation of coldness in the left side.

"In July last, while getting out of bed one morning, she had another fit, in which she became quite insensible, foamed at the mouth, and slightly clenched her hands. She remained insensible for three or four hours; and recovered with decided paralysis of the left side, inability to protrude her tongue straight, and her face drawn to the right side. On this occasion it was three or four weeks before she recovered sufficient power to enable her to walk about.

"Last Tuesday (Sept. 14), as she was engaged in cleaning a room, she fell down suddenly, the left leg and foot seeming to give way under her; she was carried up to bed, and there she became insensible for an hour; and when she came to herself again she found she had almost completely lost the use of her left arm and leg, and that sensation was much impaired.

"Since Tuesday she has been gradually recovering the power and sensation of the left side. It is still, however, very numb, and much colder to the touch than the opposite side. There is no rigidity of the muscles, and she can distinguish two points at half an inch apart, on the back of her left hand."

This patient remained in the hospital till the 15th of November, and left it much recovered in power, but still feeble on the left side.

In this case, no one can doubt the epileptic nature of the first and second attacks. The third

seems less of the epileptic character; it rather indicates a solution of continuity of brain-fibres, and possibly a slight effusion of blood; I say a slight effusion, because, under a large one, she would scarcely have recovered her consciousness in so short a time as one hour.

Case LIX. I have seen a few times lately a gentleman, a patient of my friend Mr. Lynch, of Sudbury, who has distinct epileptic attacks, immediately following the cessation of frequent periodical attacks of gout, to which he had been for many years subject. The first epileptic attack was in April, 1852; and, since that time, he has had them, at intervals of from ten or fourteen days to six weeks. Usually before the fit he has a slight warning of its approach, in the shape of a little giddiness in the head and sinking in the stomach; he then falls unconscious, the muscles of the mouth, left arm and leg, working spasmodically. The fit lasts two or three minutes, sometimes a little longer, and it leaves him with temporary paralysis of the left side, particularly of the leg, which disappears in from fifteen to twenty minutes. The next day he appears quite well. In this case there was a small quantity of sugar in the urine.

I am informed that latterly this patient has had the fits less severely, and that the paralysis is less marked, and not so extensive as it used to be.

Case LX. I am tempted to add another case which affords a good example of an important class—namely, syphilitic epilepsy with hemiplegia, occurring in an exhausted state of the system. A gentleman, æt. 25, had had chancres a few months before, followed by a slight sore throat and syphilitic lepra. These symptoms had disappeared. For some weeks he had given himself up to the hard labour of a gay life in London. On Saturday, September 24th, 1853, he went to bed in his usual health; in the course of the night he evidently had a fit, as indicated by the disturbed bed-clothes, and by his exhausted state next morning, and by hemiplegia of the left side. This recovered slightly without any medical treatment; I saw him on the Wednesday, and found that he had still imperfect hemiplegia of the left side with relaxed muscles, deviation of the tongue to the left, face-palsy on the left, and dilated pupils. There was some periosteal tenderness of the head, and the patient was evidently much exhausted, partly by the attack, and partly by previous dissipation. He was put on a treatment to regulate his digestive organs; and he took the nitro-muriatic acid, and afterwards iron and quinine. Subsequently the iodide of potassium and citrate of iron were prescribed. This treatment was pursued in the country, under the superintendence of Mr. Bennett, of Gateshead.

At the end of January, 1854, this gentleman

presented himself to me perfectly recovered, and I have seen him also in the course of the year 1855, in excellent health. The paralysis had been of only a few days' duration.

Through the kindness of my friend and former pupil, Mr. John K. Spender, of Bath, I am enabled to add a case in which very complete anæsthesia followed the epileptic attack, the voluntary power being affected only to a slight extent. I shall give the case in Mr. Spender's words.

Case LXI. "Thomas Nowell, æt. 42, an apparently healthy, well-built man, lost a leg from an accident when a boy; and suffered from repeated attacks of acute disease (the nature of which I could not clearly make out) in the earlier part of his life.

"Last winter, he had what he calls a fit, for which he was bled largely. He quite recovered from this, and had nothing to complain of till the day on which I first saw him, June 6th, 1854. He had been tolerably well that day, with the exception of a slight headache, when, at about nine o'clock at night, without previous warning, he was seized with trembling and almost immediate insensibility, which lasted nearly an hour. On awaking from this state, in which he was reported by the attendants to have been convulsed, and to have foamed at the mouth, he was found to be labouring under almost complete anæsthesia

of the whole of the left side of the body. Volition was very little injured. When I saw him, which was almost immediately after recovering from the fit, I noticed that the arm appeared swollen, and that the superficial veins were exceedingly prominent; and he was rubbing the limb very violently with the right hand. On asking him why he did this, he replied, "to get away the deadness." He could move the left arm perfectly well, and could grasp objects with the hand almost equally as well as with the other. The speech was decidedly affected; but he had perfectly regained the possession of his senses, and there was no mental hallucination, nor anything approaching to coma. Reflex action could be induced in the lower extremity. The bowels had not been moved for two or three days; the face was hot and flushed, partly, perhaps, from his violent exertions. On persuading him to remain still for a few minutes, I counted the pulse, which numbered 100. The urine was normal in quantity, but rather high-coloured. I could obtain no evidence of either rheumatism or syphilis; *possibly* some gout, but this was uncertain. The nature of his employments precluded the hypothesis of mineral poisoning.

"I moved the bowels with a brisk cathartic, applied a small blister to the back of the neck, and in forty-eight hours he was apparently well.

"He had another attack in October, very

similar to the former. Since that time he has not been to the dispensary at all, nor have my colleagues seen anything of him."

I have thus given you examples sufficient to show, that in a clinical point of view, the distinction of a form of hemiplegia in connexion with the epileptic paroxysm is well founded. The paralysis, you observe, in all these cases, follows the epileptic paroxysm. In two instances, I have known the paralysis precede the fit; and this may have been the case in the last attack of the woman, Bone (Case LVIII.), if that were really epileptic in its nature.

I cannot doubt that many of the cases of sudden loss of consciousness, followed by hemiplegia, which are popularly and even medically described as "apoplectic fits," are of the epileptic nature. It is the success of cases of this kind under bleeding, and purging, and mercury that has emboldened so many practitioners to adopt this treatment in all paralytic cases. I think the examples quoted show that the natural tendency is for the paralysis to recover without such interference.

What is the paralysing cause in such cases as these, especially when the paralysis is so transient as to pass off in a few minutes, or even hours? Those who are so ready to explain all brain symptoms by a reference to congestion, will, of course,

find no difficulty in discovering a local congestion, which occurs at the time of the fit, and remains for a longer or shorter time after it, and this congestion compresses some part of the brain, and causes paralysis. But I cannot too strongly impress upon you, that you must not rest satisfied with such a clumsy explanation as this. It is unsatisfactory in a scientific, and dangerous in a practical point of view. The vessels of a part, all-important as they are to its nutritive and other vital actions, are, nevertheless, only secondary elements in the construction of the organ; and unless in themselves diseased, they can play only a secondary part in the production of organic or functional derangement. Congestion of blood-vessels, or hyperæmia of a part, must be an *effect* either of some disordered state of the intrinsic elements of the tissue, or of the blood, or of the forces by which the blood circulates. And a sound pathology ought to receive no other explanation of morbid phenomena, or of congestion, if it exist, but that which traces the real state of these.

If any one ascribe these hemiplegic phenomena, consequent on epilepsy, to congestion, you must then ask, first, what causes the congestion on which the fit depends. A man is apparently in good health at one moment, and the next he falls in an epileptic fit. This, you say, is due to congestion. What causes that congestion? why does

it come on so instantaneously? But this is not all: A has a fit from which he recovers speedily and apparently unscathed; B has a fit of the same kind, or even lighter, and recovers from it paralysed on one side. What is the difference between the congestion of A (supposing it to exist) and that of B? Why is the latter determined to one side more than to the other?

To enter fully into this discussion would prolong this lecture to an inconvenient length, nor would it be quite consistent with its practical character. I must be content to have given you the clue to the argument on this point, and to state briefly my own view of the case.

The phenomena of the epileptic fit depend upon a disturbed state of the nervous force in certain parts of the brain—a morbidly disturbed polarity. This may take place, under the influence of some poison, which may have an affinity for those parts, such as prussic acid, in the same way as strychnine induces an exalted polar state of the spinal cord, or from some disturbance of nutrition, which may be strictly local or sympathetic. This undue exaltation of the polar force induces, subsequently, a state of depression or exhaustion, not only in the parts primarily affected, but in parts of the brain connected with them, according to the degree of the primitive disturbance; just as undue muscular action exhausts the muscular force. The disturbing cause may operate primarily, upon parts of the

brain, more directly concerned with the phenomena of consciousness, as the hemispheres; or upon parts, which when excited may cause convulsions, as the mesocephale, the region of the tubercula quadragemina. If the former be chiefly affected, and the latter slightly or not at all, convulsions are either very slight, or do not constitute a part of the epileptic fit. If, on the other hand, the latter are chiefly and primarily disturbed, convulsions form the prominent part of the fit. Now, the exciting cause of all this disturbance generally operates equally on both sides of the brain. But it may operate more on one side than the other. It leaves behind it a more or less exhausted state of brain; which, again, will be most upon that side upon which there has been the greatest previous excitement. This state of exhaustion is very apt to continue as one of weakened nutrition, in which the brain tissue is more or less in the condition of white softening. If the parts involved in this be the convolutions, mental power, memory, perception suffer; if deeper parts, as the deeper parts of the white matter of the hemisphere, and the corpora striata and optic thalami, then we have hemiplegic paralysis.

Let me relate to you two cases in illustration of the state of brain, which is apt to co-exist with the tendency to repeated attacks of epilepsy. They show, how an atrophic state of brain may be produced by the same cause, which excites the epileptic paroxysm, followed by a paralytic state.

Case LXII. Elizabeth Tribett, æt. 38 (vol. xvi., p. 164), a married woman, of temperate habits; admitted January 27th, 1846; had always enjoyed good health. Twenty-one months ago, without warning or assignable cause, while working at a mangle, she fell down in a fit, with complete loss of consciousness, without stertor, and apparently with but little convulsion. She did not recover from this for twenty-four hours, and then she was found to be paralysed on the left side and speechless. In a fortnight she recovered from this attack, and was able to resume her work.

Six months afterwards she had another fit, the insensibility lasting only a few hours; but speech was again lost, and the left side was paralysed. From the effects of this attack she recovered only very partially and slowly, although she had been sent into the country for change of air.

She now gradually fell into a state of general paralysis, with feebleness of mental powers, and great imperfection of speech. The left side was distinctly weaker than the right. All the functions became feeble and sluggish, and bed-sores formed on the nates. The sphincters did not fulfil their office, and she sunk by slow degrees. Just previously to her death she became comatose, and was slightly convulsed. She died three months after her admission into the hospital.

The inspection of the body revealed the following appearances, indicative of a chronic enfeebled nutrition of the brain and its membranes.

The Pacchionian bodies were very large, and the arachnoid membrane was extensively opaque. The hemispheres of the brain had a flaccid, flabby appearance; the sulci between the convolutions were wide, and occupied by a considerable amount of sub-arachnoid effusion. Atheromatous spots were found in the arteries at the base of the brain, especially in the anterior parts of the basilar and middle cerebral artery. The right lateral ventricle was dilated, and contained an ounce of fluid; the foramen of Monro was large, but the left ventricle did not much exceed its natural size. The septum lucidum was remarkably thin, but firm. The corpus striatum of the right side was distinctly smaller and less prominent than that of the left; it was also softer; its grey matter was especially soft. There was a slight degree of softening of the cerebral matter of both hemispheres, and great paleness of the grey matter of the convolutions; these states were especially marked on the right side. The other organs were healthy, but exhibited a good deal of congestion in their most dependent parts.

The effusion into the right ventricle in this case was due to the shrinking of the corpus striatum, and also, perhaps, of the whole of the right hemisphere. It seems to me that effusions into the ventricles, when not caused by venous obstruction or inflammation, obey the same laws as those in the sub-arachnoid space. The corpus striatum and

optic thalamus are internal convolutions, and when they shrink, fluid occupies the space which they abandon, just as fluid accumulates over the site of a shrunk external convolution.

Case LXIII. The second case many of you will remember, as it is not long since the patient was in the hospital. Abraham Metcalfe, æt. 39, a carpenter, admitted Nov. 22nd, 1852. Nothing could be elicited from this patient, or his friends, as regards his own antecedents or his family history, to account for the occurrence of his malady. He had been always healthy till last Christmas (1851), when, while engaged at his work, he was seized with "queerness," and inability to stand, and was obliged to sit down till some one came to help him into the house. He did not lose his senses, nor was he ever unconscious, but found that he had lost the use of his right arm and leg, and the sight of his right eye, and also his speech.

He recovered from this attack so perfectly in ten or eleven weeks, as to be able to go to work again.

In April, as he was walking home, he was suddenly seized as before, with precisely the same symptoms. He recovered in a great degree from the paralysis, but never so completely as to be able to resume work.

He had a similar, although slight attack on the

14th November, with increased paralysis of the right side, and his speech was much impaired.

On his admission he was found to be generally feeble in body, and sluggish in mind; the right arm and leg were decidedly weaker than the left; and a feebleness of gait, with trembling of the limbs, indicated a general weakness of his nervous system. His speech was slow and indistinct, and he seemed to collect his thoughts with difficulty. For the last three or four months he had been in the habit of passing a large quantity of urine. Shortly before his admission he passed, according to his wife's statement, as much as eight pints in one night. The specific gravity of the urine was 1012; it contained neither albumen nor sugar.

After he had been a week in the hospital, the arm and leg recovered very much; the grasping power seemed quite to return, and he could hold things out from him steadily, and button his shirt, which he could not do before.

He continued to improve until the 31st of December, when he suddenly lost the power of the right side as before, the arm being chiefly affected, and the speech almost entirely lost for a few minutes. He went to bed, and in a few hours was as well as before the attack.

On the 8th of January he had another attack, accompanied by loss of speech, and hemiplegia of the right side. From this attack he did not seem to rally; the paralysis, which was more complete

than on any previous occasion, remained; he passed his evacuations under him, and became more and more lethargic. On the 13th of January, he was seized with a slight convulsive fit, and died in a few hours afterwards. The pupils were throughout large and sluggish, and the left a little larger than the right. The daily quantity of urine fell considerably after he had been a short time in the hospital, but still exceeded the normal amount.

On examining the head there was found an abundant sub-arachnoid fluid, with some opacity of the arachnoid membrane, that on the left side exhibiting the greatest amount of opacity. The convolutions were somewhat wasted, and the sulci large. The substance of the cerebral hemispheres was softish, and seemed soaked with fluid; and this was more marked in the left than in the right hemisphere. Both ventricles were large, and contained a considerable quantity of fluid. The left optic thalamus was larger and more flaccid than the right; nothing abnormal was discovered in the corpora striata or the other parts of the brain, excepting that they all participated in the diminished consistence and soaked appearance. The basilar and vertebral arteries were large, but free from deposit. There was no indication of either local or general congestion.

In this case the attacks, although not strictly epileptic in their nature, were undoubtedly of that character. Consciousness was impaired, though not

suspended. Whatever may have been the immediate exciting cause of these paroxysms, it or they seemed to increase, for a time, the damage done to the general nutrition of the brain, and after each succeeding attack the patient showed less rallying power.

This case resembles the preceding one in the general atrophic state of the brain, and the consequent increase of the sub-arachnoid and intra-ventricular fluids. I regret much that the brain was not microscopically examined, as we should probably have obtained more decisive evidence of the exact nature of its impaired nutrition. The greater degree of softness of the left hemisphere than of the right, and the more lax state of the left optic thalamus, sufficiently indicated that the nutrition of the left side of the brain had suffered the most, and accounted for the hemiplegia being on the right side of the body. It is very desirable that in all cases of this kind in future, not only should the two sides of the brain be compared as regards their minute structure, but also as to their specific gravity.

One word more I must add. The effusion of fluid in the ventricles in this case, and the increased quantity of that in the sub-arachnoid space, were due simply to the general wasting of the brain, and had no influence in producing the paralysis. They should be regarded, in a case like this, as the consequence of that general wasting; and, indeed, it

is only when such wasting occurs, that water in the ventricles exists at the same time with an increased quantity of sub-arachnoid fluid.*

I must here conclude this lecture, reserving the consideration of choreic and of spinal hemiplegia, until another occasion.

* I have elsewhere stated that intra-ventricular effusions and sub-arachnoid fluid do not generally co-exist. A large effusion into the lateral ventricles pushes away the sub-arachnoid fluid, doubtless into the spinal canal, or prevents its secretion. So also does any enlargement or great congestion of brain, or a tumour, or a large apoplectic clot. This statement holds good in all cases in which the brain is firm and not wasted, and the exception to it is found in cases like those above detailed (LXII. and LXIII.), where there have been during life, more or less of general paralytic symptoms and failure of cerebral function, associated with impaired cerebral nutrition. My friend, Dr. Parsey, of the Warwickshire Lunatic Asylum, has communicated to me the details of several cases of general paralysis, such as are of common occurrence in every asylum, in which fluid has existed simultaneously, within and outside the brain, along with a more or less atrophic state of the whole organ.

LECTURE XV.

Epileptic Hemiplegia—Choreic Hemiplegia—Remarks on the diagnosis of the various forms of Hemiplegia noticed in the preceding lectures—Spinal Hemiplegia.

The cases, related at the conclusion of my last lecture, serve to show what kind of mischief may be done to the brain, through the disturbance caused by the epileptic paroxysm. They are examples of an aggravated amount of disturbed nutrition in the brain, ending in general softening, and a corresponding extensive weakness of the physical and mental nervous actions.

In Cases XX. and XXI., of which I have given a detailed account in former lectures,* the attack, which ultimately terminated fatally by a considerable effusion of blood, began with an epileptic fit. In the first of these cases, the early epileptic seizures were followed by hemiplegic paralysis, from which the patient partially recovered, in the intervals between the attacks. In both, the last attack ushered in the fatal rupture of blood-vessels.

The early attacks, in these cases, many would call apoplexy, *i.e.*, coma from compressed brain.

* Vide Lecture vi., p. 105, and Lecture vii. p. 121.

But I have shown that there is no good reason for supposing that the brain is, either in whole or part, subjected to compression in such attacks. And I have called them attacks of *epileptic coma*, often associated with diseased kidney, and which may or may not be accompanied by convulsions. It is very possible, and even likely, that the fatal rupture of blood-vessels and effusion of blood may be caused by the congestion, which *follows* the impeded breathing and the struggle of the epileptic paroxysm; and these accidents are the more likely to occur, when the capillary blood-vessels are in a state of fatty degeneration.

Epilepsy in its most acute form, with one convulsive fit after another, and a comatose state prolonged for many hours, will occur without any anatomical indication of compressed brain. I have related, in my lectures on Delirium and Coma, a remarkable case, in which not only coma, but paralysis of the right half of the body, ensued without any compression of the brain. The brain was not healthy, it had shrunk somewhat, and there were congestion of its surface and of the convolutions, and a large sub-arachnoid fluid, with a small quantity of fluid in the ventricles; and the only morbid condition which seemed to explain the hemiplegia, was slight softening of the left corpus striatum. There was chronic disease of the kidneys, and in this, no doubt, as well as in some chronic disease of the arteries, originated the

depraved and imperfect nutrition of the brain, and the fatal attack of paralysis and coma.

Patients suffering from an enfeebled condition of the heart, especially that from fatty degeneration, are liable to sudden attacks of coma. These cannot certainly be apoplectic in their nature—*i.e.*, from too much blood compressing the brain, or from an effusion of blood. The examination of many of these cases shows that the latter did not exist, notwithstanding that the comatose attacks had been frequent; and there are strong reasons for believing that the want of a proper supply of blood to the brain will induce a comatose state. The erect posture, for example, is favourable to these attacks, and they are sometimes relieved by the assumption of the horizontal position, or by hanging down the head. A patient here, two or three years ago, was seized with attacks of this kind whenever he sat up in bed; and Dr. Stokes relates, that a patient of his learned to ward off such attacks, by placing himself on his hands and knees, with his head dependent. They occur likewise in cases of diseased heart, in which, from regurgitant valvular disease, or from some other cause, the blood is not expelled from that organ, either in proper quantity, or with adequate force.

A very striking case is related by Dr. Stokes, as observed by Dr. Fleming, in which the patient was liable to these pseudo-apoplectic paroxysms,

or epileptic fits, as I would call them, which came on generally at night or during sleep. The patient came out of each of these attacks perfectly paralysed on the left side, and also jaundiced. Both the hemiplegia and the jaundice would subside in a very short time; the former within a few hours after the attack, and on the following day scarcely a trace of jaundice could be seen.*

"It was found," says Dr. Stokes, "that these attacks were only to be treated by the use of stimulants. During one of them, owing to a different course having been adopted, in the absence of Dr. Fleming, the patient was brought into the most extreme state of collapse. The stimulants had been withheld and the head blistered; but even under these circumstances, so decided was the effect of stimulants, that the patient who in the morning was completely hemiplegic, was within six hours completely restored to the use of his limbs."

The lesion in this case consisted in an enormous dilatation of the left ventricle, with great enlargement of the mitral orifice, which must have ad-

* Vide p. 206 of Dr. Stokes' recent important and interesting work on "Diseases of the Heart and Aorta"—Dublin, 1853. I would suggest that the jaundice in this case was a paralytic symptom, due to a temporary paralysis of the biliary ducts—analogous to the instances of jaundice from strong mental emotion or shock to the nervous system.

mitted a very free regurgitation. It was unfortunate that permission was refused to examine the brain; but from the absence of any permanent brain symptoms, it seems certain that no decided lesion could have existed. The state of brain, in all probability, corresponded with that of the early periods of the cases of epileptic hemiplegia which I have described.

Many of the epileptic seizures, which take place, only or chiefly in the night or during sleep, in elderly persons, I have reason to believe, have some intimate connexion with a morbid condition of heart—an altered nutrition of its muscles rather than of its valves.

Sleep exercises an important influence on the respiratory function—on the generation and the exhalation of carbonic acid. Thus an altered state of blood, and a modified action of the heart, are apt to occur in sleep, and these cannot fail to affect the brain in a way which may be favourable or unfavourable, according to the previous condition of that organ.

Choreic Hemiplegia.—I shall now say a very few words on the hemiplegia which is associated with chorea. In a large proportion of cases of chorea, as I have often remarked to you, the choreic movements occur more on one side than on the other, and sometimes they will be altogether confined to one side—the child being hemiplegi-

cally affected in a very exact manner. When, in such a case, the choreic movements have wholly or in great measure subsided, the patient remains paralysed in the limbs, which were before the seat of convulsive movements. The phenomena now resemble, in many points, hemiplegia, from a decided lesion of the brain. But you will, I think, generally observe the following points of difference. First, you will often find the face not affected, or if it be, only slightly so; secondly, there will not be any paralysis of the tongue, but more or less of the peculiar mode of protrusion which characterizes chorea; thirdly, the paralysed limbs will exhibit, even in a very slight degree, the choreic movements.

Case LXIV. A boy nine years of age was brought to me in December, 1848, with well-marked signs of hemiplegia of the left side; he dragged the leg and had but very feeble power of the arm; the muscles were quite lax. The face was slightly paralysed. He protruded the tongue with the choreic thrust. I learned that he had been the subject of chorea, affecting chiefly the left side for a few weeks. The choreic movements still existed, but to a very slight degree.

The patient was treated by citrate of iron, shower-baths, and exercise of the limbs, and in eight days he returned nearly well. He recovered perfectly.

Case LXV. Charlotte Plater, æt. 18 (vol. xlii., p. 239), a sallow-looking maid-servant, was admitted into Augusta ward on November 8th, 1854, with highly developed symptoms of chorea, affecting the right side of the body only. Neither the patient nor her friends could assign any cause for the attack, except, perhaps, that her daily work had been very hard, and that her food had been poor and scanty. The catamenia appeared regularly, but she had been suffering from leucorrhœa for six months prior to her admission into the hospital. After being in the hospital a day or two, the choreic symptoms almost disappeared, but left behind a paralysed condition of the muscles of the right side of the body. The account of this, given by my clinical clerk, Mr. Teale, is as follows:—"The chorea seems in great measure to have taken its departure, but has left behind it hemiplegia of the right side; there is scarcely any jerking of the muscles of the face or of the tongue, but, in walking, she appears *uneasy* in her right foot; her right arm is paralysed, her grasp with the right hand is very feeble, and she has great difficulty in picking up a pin; she complains of numbness of the right arm, hand, and shoulder, but not of the right leg or foot."

The treatment to which this patient was subjected was of the supporting kind, consisting of a generous diet, together with small doses of steel three times daily, under which her general health

gradually improved, and the hemiplegia pretty rapidly disappeared; so that, on December 16th, *i.e.*, about five weeks after her admission into the hospital, she was discharged, with the full use of all her limbs.

I may mention, in connexion with this case, that the patient was, at the commencement of the treatment, ordered a *splash-bath* every morning; but, as indeed in practice you will find every now and then happens, the first application of the cold water produced so much fright that it was considered inexpedient to repeat it.

Case LXVI. Another case, illustrative of this form of hemiplegia, is that of a little boy, named Henry Windred, who was admitted into the hospital on October 8th, 1853 (vol. xliii., p. 82). This child, who was five years of age, had always been healthy up to twelve months before his admission, when he was taken, in his mother's words, "with a trembling motion of both arms," but whether the legs were affected in the same way or not there is no evidence to show. These trembling movements continued three months, without getting materially better or worse, when, one morning, whilst being washed, he became suddenly hemiplegic on the right side, the palsy being unaccompanied by any loss of consciousness. Two or three days subsequently to this attack, the muscles of the right side of the body became

affected with the irregular jerking movements of chorea, and in this state he came under our observation. He was at once treated by upholding the general nutrition of the body in every way, and he gradually improved, the choreic movements became less and less, he slowly regained the use of his right side, and was discharged from the hospital on November 17th, about five weeks after his admission.

It is needless to multiply cases of this kind. They are of daily occurrence in hospital practice in a more or less perfect form. The case which I have related may be regarded as a more exquisite example of the paralysis.

This form of hemiplegia is liable to be confounded with one, which sometimes occurs in children from tubercular disease of the brain, and the more so as the latter form is very apt to commence with jerking movements of the arm or leg, or of both, on the side opposite to the seat of the tubercular deposit. The two conditions are to be distinguished by the absence, in the tubercular form, of the more decided chorea symptoms, and of the peculiar thrust of the tongue; by the existence, generally, of pain in the head, and of a scrofulous diathesis; by more or less of a constitutional disturbance, vomiting, fever, and general debility; and by the occurrence sometimes of general convulsions.

Now you will ask what is the actual condition

of the nerves and nervous centres upon which choreic hemiplegia depends? I answer, that it is very analogous to that which I have already explained in speaking of epileptic hemiplegia. Chorea being due to a disturbed nutrition of some part of the brain in intimate connexion with the centre of volition, the disturbing cause may act exclusively on one side of the brain, or it may operate more on one side than the other. The effect of this disturbance is first manifested in an irritative state, creating the choreic movements, and this passes sooner or later into an exhausted or paralytic state.

Opportunities for post-mortem examination after chorea, and especially the partial form to which I am now referring, are extremely rare. I have not had one of this latter form in my own experience. When such occur, the various parts of the brain constituting the centre of volition and the centre of emotion, the corpora quadrigemina and the superior part of the mesocephale, should be most diligently searched, and it is very desirable that the specific gravity of corresponding parts on opposite sides should be ascertained.

An interesting investigation of the brain in a case of general chorea, has been recorded by Dr. Aitken, in the *Glasgow Medical Journal*.* He

* *Contributions to Pathology*, by William Aitken, M.D. *Glasgow Medical Journal*, No. I.

noted the important fact, that the specific gravity of the corpus striatum and optic thalamus on the right side was 1·025, and that of the same parts on the left side was 1·031.* Further observations on this subject are greatly needed, and if made with the same minuteness and exactness as those of Dr. Aitken, will no doubt throw great light on the pathology of chorea and other allied affections.

I must bring my remarks on these forms of hemiplegia to a close, with some allusion to their diagnosis, prognosis, and treatment.

To distinguish the different forms of hemiplegia which I have brought before you from each other, is not a matter of much difficulty. Having carefully noted the actual phenomena presented by the patient, you will derive great aid in determining the particular form, to which to refer his case, from the previous history of the mode of access of the present attack. You should also investigate the patient's constitution, and inquire into his family history. It is important to learn as much as possible respecting his habits. Fail not, likewise, to ascertain the state of his kidneys, by a careful examination of the urine. I cannot too strongly impress upon you the importance of this latter point. In every case of nervous affec-

* According to Dr. Bucknill's observations, the average specific gravity of healthy brain is 1·036; according to those of Dr. Sankey, the grey matter afforded a specific gravity of 1·034; the white matter 1·041.

tion, this should be among your first inquiries. How many cases, formerly supposed to be anomalous, are now readily understood by reference to uræmic poisoning, through inefficient kidneys! Moreover, there are many other points of interest in connexion with the state of the urine in brain disease, which can only be settled by many observers; such, for instance, as the presence of sugar,—not uncommon in epilepsy,—the variations of the phosphates, the quantity of the sulphates and the chlorides, and whether, in the marked increase or decrease of these salts as elements of the urinary excretion, we can derive trustworthy aid to determine the inflammatory or the non-inflammatory nature of the brain-lesion.

A case of hemiplegia presenting itself, you may ascribe it to a special lesion of the brain, in a large proportion of cases, provided the face and tongue be affected in the usual way, on the same side as the limbs. This opinion will be confirmed if you find sensibility impaired on that side—if there be or have been pain in the head as a marked symptom, on the side of the head (or towards it), opposite to the paralysis; and if there have been premonitory symptoms prior to the paralytic attack, such as pain, cramps, numbness, or convulsive jerks in either of the affected limbs. A dilated state of one pupil, generally that on the opposite side to the palsy, is also a symptom,

which should excite your fear for cerebral lesion. The sudden invasion of the paralysis would be, on the whole, confirmatory of this view, although, in certain forms of brain-lesion, the palsy may come on gradually. In most instances, the suddenly developed palsy indicates, as I think you will find, solution of continuity of brain-fibres, either alone, or caused by rupture of blood-vessels. The completeness of the paralysis is in favour of the existence of brain-lesion, which is irritative when you find the muscles contracted and rigid from the time of the seizure; atrophic or non-irritative if they be flaccid. A suspension of consciousness may or may not occur in connexion with the paralysis of brain-lesion, as is abundantly exemplified in the cases detailed in the preceding lectures. But a sudden suspension of consciousness, whether with or without paralysis, should be always regarded by you as among the gravest of symptoms, as certainly indicating brain-disturbance, either primary or secondary.

When after a sudden attack of suspended consciousness, with or without convulsions, sometimes indeed with no more evidence of the latter than a bitten tongue, the patient comes to himself pretty soon, with a hemiplegic paralysis, but this goes off in no very long time, to be succeeded after an interval of complete or nearly complete recovery, by an attack precisely similar, such a case is a typical instance of what I have designated the

Epileptic Hemiplegia.* In investigating the history of cases of this kind it is important that you

* The following remarkable case has occurred to me while the pages of the first edition of this work were passing through the press:—

Case LXVII. A gentleman, æt. 67, of very full plethoric habit, was sitting at his club, writing letters, and had nearly completed a fourth letter, when he fell from his chair to the left side. He was taken up semi-comatose and sleepy, and was immediately attended by the late Mr. Warren Fincham. I saw him within half an hour of the attack, and found complete paralysis, with relaxed muscles, on the left side, and well-marked face and tongue palsy on the same side. He continued in a sleepy state for two hours, but would answer questions when addressed loudly. For three-quarters of an hour after the attack there was difficulty of deglutition, and imperfect ptosis of the left upper lid. The pulse was slow and irregular, and the heart's action weak. Neither Mr. Fincham nor I thought that the state of the heart's action warranted depletion.

He was subjected, therefore, to no other medical treatment than placing him nearly horizontally, and giving occasionally small quantities of water, and now and then a little brandy-and-water.

After two hours the sleepiness and stupor began to pass off, and he showed signs of recovering the power of moving the leg and arm, and in another hour he was able to grasp well, and to move about his leg freely, and to stand. In half an hour more he walked out of the club to a cab, and after he had got into bed he grasped my hand with nearly as much power as he could exert with the right hand.

He was now freely purged, and took small doses of ammonia through the night. The next day there was no

should inquire particularly respecting the previous existence of syphilis.* Contamination by this poison is very frequently at the root of the epileptic condition, and of the cerebral disease which attends it, either as cause or effect. (Case LXIII.)

To determine the nature of the cerebral lesion involves the discussion of the following questions: 1. Is the cause of the palsy a cerebral softening? and, if so, is this inflammatory or non-inflammatory; does it depend on some previous chronic disease of the brain, as a tumour, which may be benign or malignant, or a scrofulous deposit, or meningeal disease? 2. Is the lesion an apoplectic

remnant of paralysis, except in the left cheek, and slight external strabismus of the left eye. This disappeared on the following day. On the third day he seemed quite well, the face and tongue palsy having disappeared likewise; and he walked about his room with his usual strength and gait.

The phenomena of this case accord best with those of Epileptic Hemiplegia. The attack was epileptic, and probably associated with incipient white softening of the right hemisphere, from fatty degeneration of the capillaries. A premonitory symptom of this condition had occurred ten or twelve days before, in a violent cramp of the left gastrocnemius muscle, which caused considerable subcutaneous ecchymosis.

* There is a peculiar class of cases of epileptic hemiplegia, in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech. These I propose to discuss in a future lecture.

clot, which has torn up more or less of brain-substance? 3. Is the paralysis due to a compression of the brain, by serous or exudation deposit, or blood on its surface? 4. Are the symptoms in any way dependent on disease of the kidneys?

I cannot attempt to answer these questions fully in the present lecture, and must limit myself to directing your attention to one or two points, which will materially aid you in the solution of them.

First, the co-existence of disease of the heart, especially an enfeebled state of the muscular structure or of valvular disease, whether obstructive or regurgitant, with hemiplegia, is very favourable to the diagnosis of non-inflammatory softening, and, if signs of compression exist, of apoplectic effusion. Secondly, in the vast majority of cases of hemiplegia occurring after fifty years of age, which are accompanied by evidence of diseased arteries and an arcus senilis, you will have strong reason to suspect white softening, and (if other symptoms tally) consequent apoplectic effusion. Thirdly, an early impairment of consciousness will, in a large number of instances, indicate sanguineous effusion, and the existence of slight early rigidity of some of the paralysed muscles will confirm this. The comatose affection will be the greater the nearer the clot is to the pons Varolii and medulla oblongata, and the more the brain is compressed and the greater the quantity

of blood effused. Fourthly, the sudden or rapid development of coma is in general due to an apoplectic effusion. The more gradual development of it is dependent on an inflammatory softening, or an intraventricular, serous, or sero-purulent effusion, or on an exudative effusion on the surface of the brain, or on renal disease. But when you have to deal with a case of coma, you will not lose sight of the fact that it may be epileptic; and in adopting or rejecting such a view, you must be guided by the mode of access of the attack, and the previous history of the patient.

With reference to the prognosis, I would give you this advice:—"In all cases of paralysis, but especially in cases of hemiplegia, be particularly cautious in giving a prognosis. First, because it is often very difficult to form an accurate opinion as to the precise nature of the lesion, although we can generally tell whether there be lesion or not. Secondly, because few persons who are hemiplegic ever completely recover; for if once the brain be so damaged that hemiplegia results, it is very uncertain whether the powers of life are sufficient to restore or repair the injured parts; and a fresh attack, or consequent epileptic fits, are to be apprehended. The form of hemiplegia, dependent on lesion of the brain, which offers the best prospects for recovery, is that kind of white softening (if not of great extent) which occurs in young and other-

wise tolerably healthy subjects, if the brain recover its nutrition by the restoration of the circulation, the obstacle to the free flow of blood having been overcome, or a sufficient collateral circulation having been established.

The *hysterical hemiplegia* also promises ultimate recovery, but this is often very slow; and the same may be said of the *emotional hemiplegia*. In that form of hemiplegia, which is associated with *epileptic fits*, the prognosis is not in general satisfactory. The fact of the occurrence of hemiplegia shows that the brain has suffered more from the fit than is usual. It is, in my experience, very unfavourable in hemiplegia generally, when total loss of speech precedes, or occurs simultaneously with, the attack of paralysis. These cases, although the disease may not immediately prove fatal and destructive to life, seldom perfectly recover the paralysis, and still more rarely regain the power of speech. That fine old man, Ryan, now in Fisk ward, is an instance of this nature. He had an attack of hemiplegia of a slight kind, but with complete loss of speech: the hemiplegia has recovered, but although his intelligence appears good, he makes no progress in speaking. If time allowed, I could adduce several other cases demonstrating the accuracy of this statement.

Now as to Treatment.—Looking over all the forms of hemiplegic paralysis which I have described, the antiphlogistic plan of treatment is

strictly and fairly applicable only to that, in which the rigidity of the paralysed muscles occurs early, and it must then be employed proportionately to the strength and age of the patient; but recourse to *large* bleedings is certainly not justifiable in any case with which I am acquainted. By a large bleeding I mean such as exceeds ten or twelve ounces taken at once. The remedies to be used in such a case are mercury, free purging, and general or topical bleeding; but in the other forms of hemiplegia, no extensive antiphlogistic treatment should be adopted, and especially in the simple hemiplegia without loss of consciousness, which is purely an atrophic disease. You should adjust the diet to the powers of the stomach, keep the patient in the horizontal position, as quiet as possible; and carefully guard against all causes of mental agitation or excitement.

Many advocate very much the use of mercury in hemiplegic cases. It is a valuable purgative, perhaps the most valuable we possess, and as such it may be used in nearly all the forms. But for its specific influence, it ought only to be employed where cerebral inflammation exists, either primarily or around a clot, or where there has been a syphilitic taint. To use it with this object in the atrophic hemiplegia, or in apoplexy, appears to me to be worse than useless. Its employment in this way is also to be especially avoided, when there is renal disease and albuminous urine.

Then, with respect to the treatment of the paralysed limbs, some have strongly recommended the use of *strychnia,* while others have as strongly advocated the employment of *electricity;* but when the paralysis is the result of cerebral lesion, neither of these remedies promises much good, and they very frequently do harm. In the administration of strychnia, the greatest caution must be used; and if electricity be employed, it should be of feeble intensity.

There is one curious fact with respect to the exhibition of strychnia in these cases, which was first noticed by Fouquier: it is, that this agent first shows its effects on the paralysed limbs. This fact was some time ago brought forward by Dr. Hall, to show that in paralysis dependent on cerebral lesion, the irritability of the paralysed muscles is augmented. Sufficient facts have, however, now been collected to prove that this statement is not correct—that the muscular irritability in such cases is not increased, though in certain instances the paralysed muscles may be more excited by a galvanic stimulus than the sound ones. The reason why strychnia first manifests its action on the paralysed limbs is, because it is attracted in greater quantity to the diseased side of the brain than to the healthy side, and it there excites an irritative condition, which is propagated to the paralysed muscles; and this ought to point out that the use of strychnia in these cases is by no

means devoid of danger, as it tends to produce at least an irritated, if not an inflammatory, condition of brain around the seat of lesion.

The chief points, then, to be attended to in the treatment of hemiplegia, are the careful employment of ordinary hygienic measures, and the promoting the nutrition of the paralysed limbs, by moving them by mechanical means.

An affection of the bladder sometimes occurs in cases of hemiplegia, which causes much trouble to the patient and anxiety to the physician. I have seen it most developed in men of gouty constitution, and have thought it in some degree influenced by gout. It consists mainly in an urgent desire to pass water without a corresponding readiness of expulsive power. As far as I can determine, it is not influenced materially by the quality of the urine, excepting so far as that a highly acid or a highly alkaline state of the urine augments the distress. It will occur with urine of the highest and the lowest density.

The best remedies for this state are the tincture of cantharides, the cold *douche* over the region of the bladder, the dilute nitric acid carried to a high dose, and narcotics introduced by the rectum.

I shall now call your attention to Spinal Hemiplegia, a rare form of hemiplegic paralysis.

It needs but a very elementary knowledge of

the anatomy of the spinal cord to show that hemiplegia from disease of that organ can only occur when the paralysing lesion is seated high up in the cord just below the decussation of the anterior pyramids, and where it is very exactly limited to one half the cord,—*i.e.*, to one side of the median fissure.

The following case will afford you a very perfect example of the affection, and explain to you, better than any general description, the clinical history of this form of hemiplegia.

Case LXVIII. Marianne Catlin, æt. 16, admitted November 18th, 1849, had been always a delicate child, and suffered from headaches; she was pale and thin. On her admission, she complained of pains in the head, not fixed, and some stiffness of the neck. There was complete paralysis of the left arm, with flaccidity, and some wasting of the muscles. In walking, she dragged her left leg with a sweeping movement. The case had at first very much the appearance of hysterical paralysis; and this view of its nature was favoured by the absence of all palsy of the face and tongue. She was of delicate frame, and the catamenial function had been suspended for six months. She complained of a pain at the vertex, which was just that to which hysterical women are very subject. In organic disease, in women, symptoms purely hysterical are often co-

incident even in a prominent form, and embarrass the practitioner not a little. Although the palsy of the arm was complete, as regards motion, she retained some power of the leg, and could move it slowly up and down, as she lay in bed. But she was unable to hold it up from the bed. Sensibility was impaired, although she retained a considerable amount of feeling in the paralysed side.

On further examination of this patient, our attention was arrested by a marked stiffness of her neck, in consequence of which the head was drawn to the right side, and the face looked forward, and rather to the left. There was a very rigid state of the right sterno-mastoid muscle, such as one commonly sees in ordinary cases of wry-neck. On the left side the neck presented considerable deep-seated swelling in the region of the uppermost cervical vertebræ. It was evident to the touch that this swelling was not due to any accumulation of fluid, nor were the integuments and muscles in any way diseased. They were stretched over the swelling, and a thickened state of bone and ligamentous tissue could be felt through them. The rotatory and other movements of the head were much impeded; those to the right existing only to a slight degree; those to the left being limited by some mechanical hitch connected with the vertebral articulations. The patient complained very much of pain in this part

of the neck, both when pressure was made upon it and at other times; it was especially painful at night, and she found it difficult to obtain a position of ease in which to lay her head. Her nights were consequently much disturbed.

On being questioned as to the origin of this affection of the neck, she stated that it had commenced six months before her admission by stiffness and pain, the head being drawn to the right side. Soon afterwards it began to swell, and as the swelling increased, she suffered much more pain. The palsy of the arm appears to have come on gradually, as she was unable to fix the precise date of its occurrence; but it was certain that it had come on subsequently to the appearance of the swelling. She first noticed weakness of the leg on the 2nd of November, a fortnight before her admission, and this had gradually increased ever since. She attributed the pain and swelling of the neck to a cold, and denied that she had ever received a blow there.

The distinct limitation of the paralysis in this case to one side, excited our interest very much; and we discussed the various causes which could have produced it. Was it cerebral? Was it hysterical? Could any connexion exist between it and the cervical swelling? Excepting the pains in the head, which seemed very much of the same nature as those in the neck, there was no other cerebral symptom but the palsy, and this differed from that

usually dependent on brain lesion, in the absence of any affection of the face and tongue. The hysterical constitution was scarcely sufficiently developed to lead us to regard it as hysterical hemiplegia. But we found, in the pain and swelling, and the impeded motion of the neck, and the paralysis coming on subsequently to, and increasing *pari passû* with, the cervical enlargement, quite enough to explain the phenomena. I, therefore, expressed the opinion that the paralytic symptoms were due to a disease of the spinal column; that an enlarged odontoid process, or some other swelling at the upper part of the spine, had been for some time slowly compressing one-half of the spinal cord just below the decussation of the anterior pyramids; and that if the disease were not arrested, death would result from a gradual process of "pithing" by the extension of the compression to the opposite side, and by the extinction of the respiratory process. By the compression, the connexion between the medulla oblongata and the spinal cord (the seat of implantation of the phrenic and other nerves, which influence the breathing movements), would be cut off, and the mechanical acts of respiration would be rendered impossible.

From her admission to the 11th of December, our patient manifested no additional symptom of importance. She suffered much from pain in the neck and loss of rest. She was treated by iron and iodide of potassium; opiates at night; aperients

when necessary; and counter-irritation by iodine paint, and afterwards opiate applications to the neck. A generous diet was allowed her.

On the 11th of December, my clinical clerk, Mr. T. C. Dickinson, on visiting her in the morning, found that she had lost the use of the left leg completely, and that she was totally unable to do anything for herself. She complained also of twitching on the left side of her body. At three o'clock, p.m., on the same day, I saw her, and found complete paralysis, not only of the left lower extremity, but also of the intercostal and abdominal muscles of the left side, which remained perfectly motionless during respiration.* A remarkable sign indicated the want of equilibrium between the right and left abdominal muscles in expiration; the umbilicus at each expiration was distinctly drawn to the right side. Sensibility was not more impaired in the paralysed limbs than on her admission. No difference could be observed between the two sides as regards temperature. The pulse, which hitherto had not exceeded 100, now rose to 120.

On the 12th, after a restless night, the right arm was found to be partially paralysed; she was

* I am disposed to regard this latter paralysis as specially diagnostic of spinal hemiplegia. In the most complete cerebral hemiplegia these muscles are rarely affected. Vide p. 169.

just able to flex and extend the fingers slowly and feebly. The power of the sphincters was lost, and the fæces and urine were passed involuntarily. On examining the respiratory movements, it was found that the action of the diaphragm on inspiration was so feeble, that it could scarcely be felt in its descent. No contraction could be detected in the intercostal muscles of the left side, and the very slight action of that side of the chest seemed to depend on the movements of the right side. The respiratory murmur in the left lung was distinctly more audible than that in the right, especially in front. The heart's sounds were quite normal. Pulse 120, respirations 32.

On the 13th, she appeared to suffer more pain in the neck, and the least movement increased it so much as to make her cry out. Sensation was more impaired in the left arm and leg, and she complained of a want of power over the right leg. Pulse 112, respirations 32.

On the 14th, the following note was made:—

$9\frac{1}{4}$ P.M. The paralysis continues to increase. It is complete of the whole of the left side, below the neck, and of the right arm. Voluntary power over the right leg is rapidly failing; she can but just move the toes and contract the muscles of the calf, but not sufficiently to move the foot. No reflex movements can be excited. Respiratory movement seems to be performed exclusively by the right side, and feebly by the diaphragm.

Complains of great pain in the neck when it is moved. Pulse 120, respirations 32.

A few minutes after this report was made, Mr. Steele, the house-physician, was called to her, and found her speechless, with livid face and purple lips, breathing in gasps at intervals of twenty seconds. The only muscle which could be observed acting, was the sterno-mastoid of the right side; there was no perceptible motion of the ribs of either side; no abdominal movement; pulse full, 90; heart's action good at first; it afterwards, however, gradually became slower and more feeble; the pulse ceased at the wrist, and in twenty minutes the beating and sounds of the heart could no longer be distinguished. Some breathing was perceptible for a minute or two afterwards, and she ceased to exist. It is impossible to conceive a more easy or gradual mode of cutting the thread of life.

It was not a little remarkable how small an amount of disease was capable of producing such dire results. The disease consisted chiefly in an enlargement of the odontoid process of the second vertebra. This extended backwards, wearing through the dura mater, and it was covered at its upper part, and on the left side, by a fibro-cartilaginous growth, which compressed and flattened the spinal cord on the left of the median fissure. The compression of the cord was so great, that it seemed as if a large portion of the nervous matter

had been pushed from the left to the right side and partly upwards; and the cord was swollen both above and to the right of the compressed part. The nervous matter on the left side was soft and slightly discoloured, as from small ecchymoses; that, on the right of the fissure, was very soft and diffluent. The pia mater of this portion of the cord was red and congested. No other disease was found in any part of the body. It was plain, then, that the left hemiplegia, which first arrested attention in this most interesting case was caused by the compression of the left half of the spinal cord, just below the decussation of the anterior pyramids, as the pressure became gradually more and more complete. The extension of the paralysis to the right side was due to the softened and diffluent condition of the right half of the cord, a state of imperfect nutrition which no doubt was, likewise, dependent on the pressure exercised by the enlarged odontoid process.

Did time permit, I might occupy hours in discussing the many interesting points in physiology suggested by this case. The striking illustration which it affords by contrast to the law of crossed influence in brain disease; the interesting natural experiment, which it showed, to prove how the association of brain and cord is necessary to voluntary actions; and how the severance of but a small link in the chain, notwithstanding that nearly the whole spinal cord was intact, is suffi-

cient to destroy these, and ultimately other actions; the proof which it yields that but a small bond of union is necessary to preserve sensibility, if not intact, at least in a very good state.*

I shall add the particulars of a patient who recovered very much under treatment.

Case LXIX. Daniel Bryant, æt. 24 (vol. xliii., p. 130), a bricklayer's labourer, admitted November 14th. This man appears to have been healthy all his life, and free from syphilitic taint as far as could be discovered.

For five or six weeks prior to his admission into the hospital he had suffered from a pain in the back of the neck, which prevented him from throwing his head back, and greatly impeded its motion from side to side. For this he was treated as an out-patient, without improvement. On the 4th of November, after a sudden feeling of weakness, which led him to sit down for a minute or two, he found a partial loss of sensation and motion in the left arm and leg: he dragged the leg, and both it and the arm felt colder than those of the right side.

On his admission, there was partial paralysis of

* A case resembling this in several particulars has been recorded by Dr. Bright—viz. Case CXCVII., vol. ii., Part 1, of Bright's Reports. I find a more perfect example, as regards the hemiplegic paralysis, recorded by Dr. Cathcart Lees, in the Transactions of the Pathological Society of Dublin, Dec. 1843.—*Dublin Journal*, Feb., 1846, p. 221.

motion in the left arm and leg; he dragged the leg in walking; he did not admit any defect of sensibility in the leg, although there still existed some in the arm. Reflex action good. There was no symptom of brain affection, except a slight difference in the size of the pupils.

The motions of the neck were very limited, and the patient always presented an appearance of great stiffness in that region. The lateral motions of the neck were much impeded; and a decided fulness and thickening were felt in the upper cervical vertebræ. This swelling and enlargement were most conspicuous on the left side, as if in that region the greatest alteration in the condition of the bones or ligamentary textures had taken place.

The relative sensibility of the two sides was tested by points of compasses, and it was found that two points were much more readily appreciated on the right than on the left side.

On passing the galvanic current through both arms, due attention being paid to the direction of the current, it was found that the muscles of the left arm responded less readily to the galvanic stimulus than those of the right.

Our attention was also directed to the movements of the chest, as it was reasonable to expect that a compression of the cervical regions of the spine, sufficient to cause weakness of the leg, would affect these movements more or less. It was found that the breathing was less loud on the left side;

and that the movements of the ribs were less marked on that side.

The diagnosis in this case was between cerebral and spinal hemiplegia; and, in adopting the latter, we were influenced mainly by the absence of face palsy, and the existence of what seemed to be a considerable thickening (probably rheumatic) of the ligamentous structures of the upper cervical vertebræ, especially of the left side. Nor was there any symptom, except the palsy, indicative of brain disease.

The patient had a long sojourn in the hospital, during which he was treated chiefly by iodide of potassium. Four grains of this salt dissolved in an ounce and a half of distilled water were given three times a day; after a few days the dose was increased to ten grains, and the back of the neck and head was ordered to be painted with iodine paint.

This treatment was begun on the 13th of November. On the 28th of November it was reported that the fulness of the neck was very much diminished, and the movements much more free. A corresponding improvement took place in the condition of the limbs, especially of the leg.

The change for the better continued until the 19th of December, when from some cause (perhaps an increase in the rheumatic condition consequent on change of diet, as he had been allowed full diet ten days before), increased pain and stiffness, with

swelling of the neck, came on, and some diminution of power in the leg and arm. A blister was now ordered to the nape of the neck, and five grains of blue pill to be taken night and morning; the iodide of potassium was omitted; and fifteen grains of the mercurial ointment were to be rubbed in over the back night and morning.

On the 3rd of January, free salivation had been established; the neck was much better, it could be moved without pain. All medicines were discontinued for some days. On the 18th, as no further diminution had taken place in the cervical swelling, a seton was introduced at the back of the neck, and the syrup of the iodide of iron was directed to be taken in doses of one drachm three times a day. Under this treatment a gradual improvement took place; and on the 21st of February he left the hospital much better in every respect.

It is worth recording, in connexion with this case, that on three different occasions the urine was found to contain sugar in notable quantity by the test of liquor potassæ and sulphate of copper.*

* I regret much that more minute observations were not made in the case of Catlin (LXIII.) as to the state of sensibility of the paralysed limbs. It was certainly diminished, and not augmented, as Brown-Sequard's most interesting experiments would lead us to expect. In the case of Bryant, the reader will have remarked that the sensibility was diminished.

I know of no form of spinal hemiplegia, so perfect, as that which these cases illustrate. A tubercular, or other deposit, occupying with great exactness one-half of the cord; or an apoplectic clot in a similar situation, or a softening strictly limited to one side (a very unlikely occurrence), would produce a hemiplegic state on the same side of the body; but I have not met with any such cases.

It is not uncommon to meet with cases in which, from a lesion affecting one side of the spinal cord more than the other, the paralysis is greater in one limb than the other.

ADDENDUM TO LECTURE XV.

I THINK it worth while to subjoin to the case detailed in the preceding lecture, the particulars of a very remarkable case of temporary hemiplegia, which seemed to me to be of spinal origin. I give them as an extract from a clinical lecture on that, among other cases, in December, 1850.

CASE LXX. The third case on my list is that of Benjamin Mattey (vol. xxxi., p. 136), now in Fisk ward; it is very deserving of your attention in some points. He is 25 years of age. He comes from Woolwich, where he is a workman in the Arsenal. With the exception of an attack of ague four years ago, when he first went to Woolwich, he has enjoyed good health. Ten months ago, a plank fell on his left side, and struck him in the interval between the last rib and the crest of the ilium. He suffered severe pain in the lumbar region immediately after the accident, and although he was enabled to return to his work in the course of a few weeks, the pain returned, and gradually increased, and was accompanied with imperfect *paralysis of motion of the arm and leg of the left side,* with impairment of sensation. He

was now received into one of the metropolitan hospitals, where he received considerable benefit from the treatment to which he was subjected. He was freely salivated, and twice cupped. In a short time the power of sensation returned nearly completely, and he lost the pain in his back; he likewise recovered the power of his limbs in some degree. After this he was enabled to resume his work; but in nine days he was again obliged to leave off, in consequence of a recurrence of the pain. He was now placed under the care of my friend, Mr. Gallwey, surgeon in the Royal Artillery, who was then stationed at Woolwich. Mr. Gallwey established an issue in the side, and gave him iron and quinine, and cod's liver oil. Under this treatment he improved slightly, but not permanently. He was admitted into the hospital on the 14th of November, and at this time the pain had assumed to a remarkable degree a periodical character. It commenced at night when he went to bed, and lasted for three or four hours, subsiding gradually. On awaking in the morning, he would feel a soreness in the region, where the pain had existed the previous night. It was a relief to get out of bed, and assume the erect posture. But this, as all other movements of the trunk, was done with great care and caution. Any sudden contraction of the muscles of the left side caused severe pain—pain somewhat of the same description as that which a man would suffer from sud-

den muscular action, when labouring under a fit of lumbago.

A careful examination of the spine discovered nothing wrong there; but a decided pain was produced by pressure over the region of the quadratus lumborum muscle, and the act of coughing or sneezing was extremely painful.

From the fact that the pain had its seat where the injury occurred, and also because it was excited by muscular exertion, when the lumbar muscles on the left side were in action, it seemed to me clear that great part of the pain was due to mischief going on in the muscles and fasciæ of the left lumbar region—the result of the injury inflicted by the fall of the plank. Some of the muscular fibres had been probably ruptured, and perhaps also some of the nerves themselves had suffered. The periodical nature of the severe pain led to a suspicion that it might be regarded as neuralgic (the position being determined by the injury, and the periodicity by some remnant of the marsh poison, which may have remained in his system since the attack of ague), and Mr. Gallwey seems, judging by his treatment, to have taken much the same view.

But there are other symptoms of the case which do not admit of so easy an explanation: the paralysis, namely, of sensation and of motion. The parts affected are the left upper and lower extremities. I read the following account from the

case-book :—"The sensibility of the left arm is decidedly diminished, so that he cannot distinguish two points of a compass placed on it or the fore-arm, an inch apart. He is unable to grasp firmly with, or completely to extend the fingers of the left hand. The sensibility of the leg is not so much diminished, but he is not able to bear much of his weight upon it, and he walks as if his legs were tied together. There was no tenderness of the spine, and he has full power over the sphincters."

Now, the hemiplegic character of the paralytic affection would certainly seem to indicate, *primâ facie*, that it had its origin in the brain. But this view is quite untenable, seeing that he is perfectly free from every other symptom of disease of that organ. It seems to me that we can only explain his symptoms by supposing an extension of some inflammatory condition from the lumbar nerves and muscles to the dura mater of the spinal cord. The chief difficulty in the way of adopting this view arises out of the extent of the paralysis, which involves the supposition of a corresponding extent of affection of the dura mater ; and it is difficult to conceive the existence of so extensive disease of the dura mater with pain so completely limited to one spot. But if we consider the intimacy of the connexion of the dura mater of the spine with the spinal nerves, we need not scruple much to admit that a slight affection of that mem-

brane would impair the power of the nerves sufficiently to explain the existing degree of paralysis. Or it might be supposed that the injured nerves exercise some depressing influence on the region of the cord, in which they are implanted, and this, by its reflection, may cause a similar state of depression of nerves higher up. On the whole, the more probable view seems to me to be, that the injury has created an inflammatory condition of the fibrous tissues external to the spine, and that this is extending to the meninges, and perhaps even to the bones.

At first we treated this man on the supposition that the pain was purely neuralgic, and he had large doses of carbonate of iron. After three days' trial of that plan, I resolved to deal with it as a case of inflammation of a fibrous tissue, and then gave him iodide of potassium in ten-grain doses thrice a day.

The results of this treatment were extremely interesting, and very deserving of your attention.

On the 20th of November, he began to take the large doses of the iodide. A decided iodism made its appearance in a very short time. After the first dose he felt an uneasiness in his head, and after the third dose he began to feel as if he were tipsy; and a profuse discharge flowed from the nose, and the eyelids became œdematous, with a slight blush of redness; at the same time there was a flow of saliva as copious as if he had been

freely salivated. In consequence of these symptoms, he was not permitted to take a fourth dose of the medicine. During the latter part of the 21st and the 22nd, he took no medicine. On the 22nd there was evidently a considerable improvement as regards the pain; most of you will remember how distinctly the expression of his countenance indicated relief from pain. On the 23rd the iodism had completely subsided, and I now ordered the iodide of potassium to be resumed, in the same doses as before. Immediately after taking the first dose on the second occasion, there was a return of the flow of saliva to the amount of half a pint; but on continuing the use of the medicine, the ptyalism subsided, and a tolerance was established. Under this treatment he improved greatly—the severe periodical pain subsided—he is able to move about much better, and is regaining the power and sensibility of his limbs.

It is worth while to notice here a curious change in the characters of the urine, which occurred while he was under the influence of the first doses of iodine. On the 20th November the urine was pale, and the sp. gr. 1006. On the 23rd, the quantity was much as before, that is, normal, but the sp. gr. had increased to 1030, and there was a copious deposit of the lithic acid; and on the 25th, the sp. gr. was 1035; there was an abundant precipitate of lithate of ammonia,

and there was evidence of the presence of bile in the urine from the play of colours caused by the addition of nitric acid.

I presume no one can doubt that, in this case, the peculiar group of symptoms which we call iodism, the diminished pain, the increased power in the limbs, and the remarkable changes in the urine, bore to the iodide of potassium, as administered to him, the relation of effect to cause. Doubtless the iodide has promoted elimination through the kidneys and the salivary glands of some material which at least was maintaining a febrile state of his system. Can it be that the man was of a rheumatic or gouty diathesis, increased by a sedentary life since the accident, and that this kept up and increased the pain?

I fear that the favourable change will not be permanent, and is only due to the temporary clearing out he has experienced from the iodide; and I am led to this opinion by the fact, that on a former occasion he experienced nearly as great improvement from the use of mercury, but speedily relapsed.*

* The further history of this case was as follows:—The patient continued to improve up to the 11th of December, having taken the iodide (to each dose of which, on the 29th of November, five grains of citrate of iron were added) during the whole time. He then left the hospital, stating that he could walk better than ever he did since the accident. The day after he left the hospital the pain

returned, he became gradually worse, and was re-admitted January 1st, 1851. The pain was now continuous; there was spinal tenderness over the last lumbar vertebra, the pain was much increased by turning in bed or stooping; he moved with extreme caution: a sudden jar or false step was exquisitely painful. The weakness of leg and arm had returned. He was told that his symptoms indicated disease of the vertebra, that great quiet would be necessary, and that for a long time. He then proposed to leave the hospital, preferring to remain at home. He was discharged on the 10th of January, and since then it has been ascertained, through the kindness of my friend, Mr. Fogo, of the Royal Artillery, that he was discharged from the dockyard, having shown unequivocal signs of caries of some of the lumbar vertebræ, with paralysis of the lower extremities.

LECTURE XVI.

On a case of Epileptic Coma and Lead Palsy.

In Fisk ward we find a man of the name of J. Clarke, æt. 30 (vol. xxxii., p. 21), who was admitted here about two months ago, Oct. 1850.

Case LXXI. This case exhibits an example of two very interesting and highly important classes of disease—epilepsy, and the effects of lead poisoning. He was admitted in a state of profound epileptic coma; and from October the 18th to the 20th, he had five convulsive fits, accompanied and followed by coma, which continued for two days, when he began to recover.

The long continuance of a state of profound coma is always calculated to excite anxiety in the friends and attendants of a patient, and to create fear lest some extensive mischief shall have been done to the brain. In this case there were two circumstances among others which greatly encouraged me to believe that no serious lesion existed in that organ. The first of these was, that the coma was accompanied with epileptic convulsions. Now you will not understand me to say that I do not apprehend danger from such pa-

roxysms of epilepsy as this man has had ; but that, when coma is followed by a succession of attacks of epileptic convulsions, and when there is no hemiplegic paralysis, we have a good deal of presumptive evidence that there is no apoplectic clot, or other organic lesion likely to damage the brain permanently. Thus we were led to ascribe both the coma and epilepsy, not to the pressure of a clot upon or within the brain, but probably to one and the same cause, which cause was suggested by the second circumstance to which I have referred—namely, that his urine was scanty in quantity, and highly impregnated with albumen. I viewed the case, then, as one of those in which the cerebral affection was due to the presence of some irritating matter in the blood which ought to be eliminated by the kidneys. There are very good grounds for believing that when urea is retained in the blood, the brain is very likely to be affected so as to cause coma and convulsions. Other substances retained in undue quantity may produce the same effect, for aught we know ; and certainly, coma and convulsions may occur in cases where we have no evidence of the presence of urea in the blood ; but it is quite as certain, when the kidneys fail in their action and secrete only an ounce or two of urine in a day, instead of thirty or forty ounces (whether the poisonous agent be urea or something else), that coma and convulsions are very apt to ensue.

And in such cases we get proof of the presence of urea in the blood, as we have done in this case. The following is the method which was pursued for this purpose:—A blister was applied to the back of the neck; and when it rose, the serum was carefully collected, and tested for urea. The whole quantity of serum was evaporated to dryness over a water-bath, and the residue was extracted with alcohol, which is a ready solvent of urea. This alcoholic extract was then evaporated to dryness, and a little water added so as to make a syrupy mass, which was plunged into a freezing mixture, and a few drops of pure nitric acid were added. If urea be present, the characteristic crystals of nitrite of urea are soon formed in the solution, and may be recognised either by the naked eye or by the microscope.

Our patient is an intemperate man, and filled the place of a waiter at a low tavern. We have not been able to trace any immediate exciting cause of his present attack, except exposure to cold shortly before. It is probable that, under the influence of his habits of intemperance, renal disease had been making insidious progress for some time; but, on exposure to cold, the action of the skin having been checked, an acute affection of the kidneys was induced, these organs became highly congested, their tubes filled with epithelium, allowed but a small flow of urine, already curtailed in its quantity and charged with

serum, whence its albuminous impregnation. This defective secretion of urine allows urea or some other poisonous material to accumulate in the blood, which, passing to the brain, so far disturbs the nutrition of that organ as to excite convulsions, or coma, or both.

As soon as recovery from the state of coma took place, we observed a paralytic affection of the upper extremities, and of those alone; the legs were in no way involved. Nor did the paralysis involve all the muscles of the upper extremities: those of the arm and shoulder were only very slightly affected; the muscles of the forearm were chiefly engaged, and of these the extensors were most distinctly paralysed. All were wasted; but the extensors most so, as was obvious from the hollow which existed over those muscles on the back of the forearm. His power to flex the wrist and grasp with his fingers was very feeble, but he was wholly unable to extend the wrist or the fingers. When the arm was stretched out from the trunk, the hand hung, as if lifeless, from its articulation at the wrist. Both upper extremities were affected in precisely the same way, although not precisely to the same degree; for the right forearm was evidently weaker than the left. The muscles which form the prominence of the ball of the thumb (the *thenar* eminence of surgical anatomists) were also paralysed and greatly wasted, and the power of flexion, or ex-

tension, or adduction of the thumb, was almost entirely destroyed.

It is plain that in this case there were two classes of symptoms with which we had to deal—an acute class, which yielded pretty readily to treatment, and which must have destroyed the patient sooner or later if they had not given way; and a chronic, which remained after the first had been removed, and which seemed to have no tendency to shorten life. The first were the coma and convulsions—the second, the palsy.

Viewing the first and acute class of symptoms as indicative of the state to which I have given the name *renal epileptic coma*,* I adopted a treatment actively eliminatory, with a view to remove by other channels, as much as possible, the material which was disturbing the brain. The channels through which I endeavoured to conduct this noxious matter, were the intestinal mucous membrane and the skin.

His head was shaved, and he was freely blistered over the scalp; mustard cataplasms were applied to the back of the neck, and he was very freely purged. The best purgative to use in such cases is elaterium, because it acts promptly, and produces liquid stools, carrying off large quantities of water from the system, which, no doubt,

* Lectures on Delirium and Coma. *London Medical Gazette*, 1850.

like that obtained from the blister, contained urea. Warm baths, or hot-air baths, may often be used with great advantage in these cases; but I did not allow Clarke to take more than one of each, as I thought him too weak to be subjected to remedies which, especially the latter, have a decidedly depressing influence.

He was admitted on the 18th of October; on the 22nd he was quite free from any comatose symptom, and there now remained to be dealt with the condition of the kidney (the *fons et origo* of the cerebral disturbance), and the paralytic state.

The urine had increased considerably in quantity; it was still, however, very highly albuminous, becoming almost solid by heat, and of low specific gravity; and, under the microscope, contained casts of tubes and epithelium, and some blood corpuscles. It was plain that, whatever might have been the former state of the kidney, it was *now* very much irritated, and that the defective secretion and albuminous impregnation were due to this.

The condition of the kidneys has improved greatly in this case under the further use of purgatives, at first elaterium, and afterwards compound gamboge pill, and also of warm baths; so that now the urine contains very little albumen; but the paralytic state has remained very little changed.

It exhibits precisely the character of that form of palsy which results from lead-poisoning, more commonly known as the "painter's wrist-drop." All the characteristic signs of this form of palsy were as well marked in this case as in any case I have ever seen. When you make him hold out his arms, you see both hands hang down, and he has no power to bring them to the state of extension.

A practical man could not see such a case without asking if the patient was a house-painter. Yet we found, on inquiry, that not only was he not of that trade, but that his proper vocation, that of a waiter, did not particularly expose him to the lead contamination.

Can the palsy be due to any particular lesion of the nervous centres, independently of lead? Or is it the result of the renal disease? It is not likely to be the latter, for it existed before the attack of coma, and its origin dates as far back as four or five years, and was preceded by two attacks of which he gives but a very imperfect account, but which were attended with obstructed bowels and severe pain in the belly—attacks resembling lead colic. He was at this time in Devonshire; but he states that he did not drink cider.

I do not think that the palsy can be attributed to any special lesion of the nervous centres. I know of none which would produce exactly this

form of paralysis—so symmetrical—affecting particular classes of muscles in preference to others, and those of the forearm especially, and without any damage to sensation.

We are led, then, to attribute the palsy to contamination by lead; and, in confirmation of this, we have found the blue margin to the gums. The blue line is not uninterrupted, for he has lost several teeth, and at these points it ceases; but wherever there is a tooth, or a stump projecting above the gum, there the blue line is distinct.

But the difficulty in this case has been to explain how the lead came to be introduced into the system. It is true we are exposed to this contamination from the water we use, and we shall be so, as long as there are leaden cisterns and leaden pipes to convey the water. In time it may be hoped that glass may be substituted, or some other substance not likely to yield up poisonous matter to the water. The wonder is, not that an occasional case of this kind occurs, but that they are not infinitely more frequent. Some people, no doubt, exhibit the idiosyncrasy of being strongly affected by very small quantities of particular poisons, which it is generally necessary to administer in much larger doses to produce their specific effects. We see this often in the use of mercury and of iodide of potassium. It is possible that this man may have this idiosyncrasy as regards lead.

I think we have a better explanation than this. It appears that a part of our patient's duty was to clean and keep bright the pewter pots belonging to the public-house to which he was attached. This he did by friction with his hands. Now pewter very commonly contains lead in considerable quantity; and no doubt the frequent contact of this with the hands would lead to a gradual absorption of a sufficient quantity of the metal to produce the poisonous effects; or the repeated frictions might cause the separation of minute metallic particles, which might be inhaled.

The palsy from lead is most probably due primarily to the contamination of the muscles by the lead: these structures become impaired in their nutrition, and the nerves, especially the motor nerves, suffer in consequence of their comparative inaction. Doubtless, after long exposure to the contaminating influence, the nervous matter itself will become poisoned, and thus in such cases brain symptoms ultimately show themselves. But the palsy may be regarded as a form in which the loss of motion is in the first instance due to a diseased state of the *muscles* themselves,—not, as is generally the case, to disease of the nerves, or of the nervous centres.

The theory of lead palsy, which refers it to lesion of the spinal cord or brain, evidently will not account for the phenomena. It will not account for the muscles of the forearms being

chiefly affected; nor will it explain the symmetry of the affection; nor the greater palsy and wasting of extensors than flexors; nor the special affection of the muscles of the thumb; nor, in fine, the almost total exemption of the nerves of sensation amid so much injury to the motor function. Moreover, I think it may be affirmed with perfect truth, that a lesion of the spinal cord sufficient to create so much palsy of the upper extremities, as we often see in such cases as these, would necessarily affect other parts also; whereas this man Clarke has full power of his lower limbs, his mind and senses are perfectly clear, and he has no symptom of any nervous affection besides the palsy of the upper extremities.

I have already explained to you the treatment adopted in this case for the renal disease. Fortunately, that necessary to remedy the effects of the lead poisoning is much of the same kind. He has had frequent sulphur baths, with evident advantage to his general health. I have seen many instances of great improvement to the state of lead cachexia following the long-continued use of sulphur baths.* He has also gone through a long course of galvanism with very little benefit. One arm has been fixed upon a splint, according to Dr. Pemberton's plan, while, for the sake of comparison, the other arm was left free. It was evident

* Vide Lect. i.

after this treatment that the arm which had been bound up was worse than before. On the whole, the lead palsy is very little better than on his admission; nor is it to be expected that it should be, in so short a space of time. The poison of lead damages the muscles so much, that it requires a very long time before any marked change takes place: and I do not know that any one remedy exercises a specific influence, unless it be iodide of potassium; but there can be no doubt that all those hygienic means, which contribute to promote a sound general nutrition, are the most useful in aiding the recovery of the patient.

The sequel of this case is very interesting.

The patient Clarke (vol. xxxvii.) left the hospital in January, 1851, after a sojourn there of three or four months, during which time all his symptoms improved very much, the albumen having disappeared almost completely from the urine, and the arms having become stronger; but a certain amount of weakness of the extensor muscles still remained. He did not return to his former employment, but went into the country, and he continued well until the summer of 1852, when he again applied for admission into the hospital, in consequence of increased weakness of the arms. Under the sulphur treatment he again improved, and he remained well until the 22nd November of the same year. He now began to feel very

unwell, and on the 24th was seized with violent pains in the belly, and cramps, and pains in the thighs and legs, for which he was admitted a third time on the 26th November.

These symptoms, which seemed to arise from either gouty or lead colic, were quickly subdued by free purging, warm bath, and counter-irritation by turpentine stupes applied to the surface of the belly, and a single dose of morphia, and on the 27th he was very much better. But his urine was found to be highly albuminous.

On the morning of the 29th he complained very much of swimming in the head, which rapidly got worse, and he went off into a fit of epilepsy, which lasted but a short time; during the fit the muscles of his face and limbs were slightly convulsed, the fists were clenched, the eyes were fixed in the upward direction, and he foamed at the mouth; and was quite unconscious. The urine was of sp. gr. 1015, and contained abundance of albumen. The quantity of urine passed could not be ascertained, but there was reason to believe it was below the normal amount.

The following note of the condition of the urine on the 1st of December was made by Mr. Evans. Its quantity, in the last twenty-four hours, was two pints and a half, sp. gr. 1016, pale, clear, acid, deposits a slight densish sediment, and contains a considerable amount of albumen. Its microscopic appearances are as follows:—The whole field is

covered with pus-cells chiefly agglomerated in masses; a mass of smooth, oval, flat, epithelial particles (probably from the urethra); a granular cast with one epithelial cell upon it; a large waxy cast (from a tube probably deprived of its epithelial lining); a granular cast about $\frac{1}{700}$ inch in diameter; there is no appearance of the pus being moulded.

Thus we had quite sufficient evidence in all these symptoms to indicate that, while our patient was out of the hospital, whether from exposure to cold, or intemperate and irregular living, or all combined, fresh irritation and inflammation of the kidney had come on. He was ordered the use of the hot-air bath thrice a week; his diet was regulated, and purgatives occasionally administered.

Under this treatment he improved steadily in general health and strength; the quantity of urine increased considerably, averaging three pints a day, sp. gr. 1012, and presenting much the same characters as those already described, with a very gradual diminution in the quantity of albumen. On the 31st of December, the quantity of albumen was about one-eighth of the portion examined; no cast could be seen, but there were still several pus-cells. He was now ordered small doses of the tincture of the sesquichloride of iron.

On the 15th of January, the bowels having been constipated, probably by the iron, he had a fresh attack of colic, accompanied with violent vomiting

and retching, which lasted for three days. On the 20th, another epileptic fit occurred, just like that on the 29th November, which left him very debilitated and giddy, with dim vision. From the commencement of the attack of colic, the quantity of albumen in the urine increased considerably. On the 19th, the urine became almost solid by heat, and numerous pus-cells were seen in the sediment.

On the 24th, he had a fit of the gout in both great toes; he stated that he had frequently had similar attacks of gout before, preceded by colic. Since the appearance of gout in his toes, the abdominal pain had completely ceased. The quantity of urine increased; on the 25th it was five pints and a half, the albumen still very considerable, but diminishing. On the 22nd he was ordered the bromide of potassium, in doses of three grains thrice a day.

He now gradually passed into his former state. All traces of gout disappeared. He passed urine largely, averaging four pints daily, sp. gr. 1011, its sediment containing pus-cells, some lithic acid, and casts occasionally; and on the 3rd of February the quantity of albumen had diminished to the same point as before the fit, namely, one-eighth the quantity examined.

On the 13th, however, a very marked increase having taken place in the quantity of albumen, I ordered him to have the hot-air bath twice a day,

for a few days, hoping by promoting increased action of the skin, to forestal any further attack of epilepsy.

This treatment apparently answered; the quantity of urine was now the same as the quantity of liquid drunk; and the albumen gradually diminished. On the 19th, the hot-air bath was given up, and he was ordered fifteen minims of dilute nitric acid thrice a day.

On the 23rd, a fresh attack of gout appeared, without any colic. This speedily gave way without any special treatment, and he steadily improved. On the 1st of March, 1853, he left the hospital, having gained strength, the paralytic state being much the same, and the kidneys acting as before.

I had repeatedly called the attention of the Class to this case, as affording illustration of so many morbid processes. We had, in this one patient, an example of lead-poisoning causing the peculiar palsy; we also had that which frequently accompanies the lead-contamination, namely, gout; there was also colic, which probably was gouty; there were diseased kidneys, likewise gouty; and although last, not least, there was epilepsy.

In June of 1853, Clarke was again admitted into the hospital, having been for some time treated as an out-patient, for rheumatic pains. He now resumed the use of the hot-air baths, and improved slightly under them. After having been a fortnight in the hospital, he was seized with

seven epileptic fits in rapid succession, from the exhaustion consequent upon which he died.

The post-mortem examination took place on the 18th.

The kidneys weighed each three ounces and three-quarters; their surface was slightly uneven and granular. Numerous cysts existed in them, most of which were very small, but one was as large as a walnut. Many of the tubes were atrophied and denuded; many of them were opaque with granular disintegrated epithelium. The small arteries were thickened, and their coats hypertrophied.

No pus could be discovered in the tubes or in any part of the kidney. It seems most likely, therefore, that the numerous pus-cells which were so often found in the urine, were derived from the mucous membrane of the pelvis of the kidney and of the ureter and bladder (gouty irritation). That mucous membrane, however, after death, exhibited no marks of disease.

The brain was exceedingly pale and free from blood; but in other respects, as far as the eye could determine, its characters were those of health.

In consequence of the prominence of the nervous symptoms, and the evidence of that contamination previously alluded to, I thought it desirable that the specific gravity of different parts of the brain should be carefully ascertained,

and that both it and the paralysed muscles should be examined as to their physical state, and also with reference to the presence or absence of lead. This inquiry has been most ably conducted by my friend, Dr. Conway Evans, whose report is as follows :—

"The experiments, by which the following results with respect to the specific gravity of the brain were obtained, were performed on the evening of June 18th, 1853, sixteen hours after death.

"The specific gravity of the various parts of the brain, given in the annexed table, was obtained in the usual way for determining the specific gravity of a solid, heavier than water, and insoluble in, and not chemically acted on by that liquid. A piece of brain substance was first weighed in air, and then in distilled water; and by a comparison of these weights the density of the portion of brain experimented on, in relation to water as 1000, was determined; or, in the words of the school-books, 'the weight in air was divided by the loss in water,' the quotient so obtained being the specific gravity.

"The weights of the various portions examined were in every case very accurately taken, the greatest care being used that any mistake which might unavoidably occur should be less than the one-hundredth part of a grain.

"The weights of the several portions of which the specific gravity was ascertained, varied from

56·09 to 248·8 grains, the average of the whole sixteen experiments being about 115 grains; and great pains were taken in estimating the density of similar parts on opposite sides, to use, as nearly as conveniently could be done, equal weights of corresponding parts (although in several cases the weights differed considerably), and also to remove those parts from exactly similar situations on each side of the brain.

"The following Table shows the results obtained in this manner:—

Part of Brain.	Sp. Gr. Water being 1000.
White substance of right hemisphere	1040·8
White substance of left hemisphere	1039·5
Grey matter of convolutions of right hemisphere	1035·8
Grey matter of convolutions of left hemisphere	1036·8
Right Corpus Striatum	1040·0
Left Corpus Striatum	1039·6
Right Optic Thalamus	1040·6
Left Optic Thalamus	1041·1
White and grey matter of right hemisphere of Cerebellum	1042·9
White and grey matter of left hemisphere of Cerebellum	1043·2
Right side of Pons Varolii	1039·7
Left side of Pons Varolii	1039·5
Right Crus Cerebri	1038·0
Left Crus Cerebri	1039·0
Right half of Medulla Oblongata	1037·9
Left half of Medulla Oblongata	1037·9

"The extensor muscles of both forearms, but especially those of the right, were of a very pale, almost fawn colour, and had a very wasted appearance; and on tearing, they were found to be much tougher than healthy muscle. Under the microscope they appeared to have become converted almost wholly into wavy bands of 'white-fibrous' tissue. Here and there a muscular fibre could be seen running amongst the fibrous bands, but in these the transverse striæ were very ill-defined; indeed there seemed to be hardly any transverse markings at all, and the whole fibre had a peculiar pale, faintly granular appearance. There were not the least indications of fatty degeneration. When treated with acetic acid, the whole specimen displayed vast numbers of nuclei, chiefly elongated and fusiform, their long diameter being, for the most part, in the direction of that of the fibres.

"The red, healthy muscle when submitted to microscopic examination, exhibited no departure from the normal structure.

"A portion of the diseased and healthy muscles, and also of the brain, were subjected to chemical analysis with the view of detecting the presence of lead in those tissues.

"The portion of healthy muscle experimented on weighed 774 grains, and the diseased muscle 736 grains; and the process employed, which was precisely alike for each, was as follows:—

The muscular tissue, having been thoroughly dried over a water-bath, was incinerated, the temperature being gradually raised to a very high degree. By this means a carbonaceous residue, containing all the fixed salts, was obtained, and this was carefully examined throughout to ascertain the existence of any minute metallic globules, which would, probably, have been formed, had the muscle contained lead, in consequence of the reduction of the metal by the carbon of the organic matters. No metallic globules, however, having been discovered, the carbonaceous residue was boiled with dilute nitric acid and filtered; and through the clear solution a stream of hydrosulphuric acid was passed, with the view of precipitating, in the form of sulphide, any lead which might exist in solution.

"In the case of the healthy and of the diseased muscles, the results obtained were precisely similar; for in neither case was there the slightest trace of a metallic sulphide thrown down, the only precipitate being some sulphur, resulting from the decomposition of some of the hydrosulphuric acid.

"Portions of various parts of the cerebrum and cerebellum (chiefly the white substance), weighing in all 4000 grains, were also analysed; and the process adopted was exactly similar to that employed in the examination of the muscles, except that the carbonaceous residue, obtained

by incineration, was boiled with a solution of carbonate of soda and filtered, previous to its being treated with dilute nitric acid. The object of this was to remove as much as possible of the fixed salts before getting the lead, if any were present, into solution. When the clear acid solution, obtained from the carbonaceous residue, was subjected to a stream of hydrosulphuric acid, a clotty, orange-yellow precipitate, very like in appearance the tersulphuret of antimony, was thrown down. This precipitate, when thoroughly dried, weighed 0·33 grain; but it was, most probably, nothing more than sulphur in combination with organic matter; for, when heated, globules, which, when examined under the microscope, were seen to be evidently those of sulphur, sublimed, while a carbonaceous residue remained behind."

"So far, then, as the process employed in these cases is accurate, it appears, from these analyses, that the portions neither of the healthy nor of the diseased muscles, nor of the brain, which were examined, contained lead in appreciable quantity; and inasmuch as this method is an exceedingly delicate one, it seems fair to infer that had lead, even in minute amount, been present in these tissues, it would have been detected as the black sulphide, when its solution in dilute nitric acid was subjected to a stream of hydrosulphuric acid."

We are not yet sufficiently certain as to the normal specific gravity of the brain to justify any

inference being drawn from the facts above collected on that point. What I have now recorded must remain, as a contribution to that large series, which has yet to be accumulated, before we can legitimately begin to reason upon this subject.

As to the absence of lead, this is not to be wondered at, notwithstanding the still wasted state of the muscles, when we consider how long a time had elapsed since the lead poisoning (between four and five years), during which time he had been frequently under medical treatment. Viewing the palsy as due to lead, this inference may be drawn, that the impaired nutrition previously induced by the lead contamination may not become restored even after some years. By an unfortunate oversight, the nerves of the diseased muscles were not examined.

In juxtaposition with this case of Clarke, I shall place another—in many respects very similar—illustrating how the contamination of gout generating diseased kidney may give rise to further contamination of the blood, out of which shall arise the most formidable and ultimately fatal disturbance of the brain's function. The following account of the case is taken from notes kept by my clinical clerk, Mr. C. Macnamara.

Case LXXII. Mary A. Parry, æt. 44 (vol. xlii., p. 5), admitted May 19th; a married woman

and mother of twelve children, the youngest eighteen months old; has menstruated very profusely within the last three days. Her father died of gout and dropsy; mother died young.

This woman had, on admission, a remarkably dirty sallow complexion (very common in cases of chronic renal disease); she had been losing flesh very much for some time, and was in a state of great debility. Ever since her twenty-fourth year, she had been subject to attacks of gout. Almost every year she has had an attack, which sometimes lasted only a week, sometimes five or six weeks. It affected both large and small joints of the extremities, and has left deposits in the tendons and ligaments of the finger-joints. For some time past her breathing has been getting short, especially upon exertion.

The illness, on account of which she sought admission into the hospital, came on five weeks before with increased dyspnœa, and an attack of gout in her ankles. The gouty attack yielded, but the ankles remained swelled, and from that time her lower extremities and abdomen quickly became dropsical: and the quantity of urine passed, which previously had been considerable, gradually dwindled down to only a pint in twenty-four hours. The whole of the lower extremities were œdematous, and the integuments of the abdomen and of the loins were in a similar state; and a considerable quantity of water had accumu-

lated in the peritoneum, causing great distension of the abdomen. The urine was loaded with albumen. Pulse very small and weak; heart's sounds feeble and distant, with a very distinct mitral systolic bellows sound. The superficial jugular veins were large and distended, and when compressed in the neck, did not empty themselves readily on the cardiac side of the point of pressure. She had cough with free mucous expectoration, and a good deal of crepitation was heard throughout both lungs.

Upon microscopical examination by Mr. (now Dr.) Conway Evans, the urine was found to contain crystals in such abundance, that everything else which composed the sediment, was almost totally obscured; here and there, however, a large and a small waxy cast were seen. The crystals were chiefly thick, yellowish lozenges (uric acid), and large, irregular-shaped masses, apparently consisting of the above-mentioned lozenge-shaped crystals aggregated together. With these there were many of the masses provided with projecting needle-like processes, generally regarded as urate of soda, and also many colourless rectangular prisms; also a good deal of broad pavement-epithelium probably from the vagina.

The case was regarded as one of gout, with gouty kidney, upon which acute inflammation had supervened. To this was added disease of the heart consisting of imperfection of the mitral

valve, probably from shrinking of the chordæ tendineæ and the fibrous basis of the valve, with dilatation of the ventricles, especially that of the right side; œdema of the lungs; congestion of the liver, consequent on the feeble action of the heart. As a distinct grating was felt on moving many of the small joints of the fingers and toes, it was inferred that their cartilages were replaced by gouty deposit, and it was conjectured that a deposit of the same nature damaged the valves of the heart.

Under the use of diuretics and sudorifics, the quantity of urine increased to upwards of a quart for the first ten days, the albumen being still very abundant, and the belly diminished in size. On the 30th, a diminution took place to a pint and a half; the pulse rose from 100 to 120, she had passed a bad night, and she complained of being much troubled with convulsive movements of the arms and legs.

On the 31st, at six in the morning, she got out of bed and sat for a short time before the fire, as if she had been chilly. Soon after getting into bed, she was suddenly seized with a fit, in which the whole body was convulsed, she foamed at the mouth, and the tongue was protruded between the teeth and bitten. This was followed by several other fits, each lasting about ten minutes, leaving her in a state of extreme prostration and exhaustion, and apparently insensible,

until another fit was about to come on, when she would get up in bed and answer questions; and expressed herself as feeling very well and free from pain. Between the fits the pupils remained very much contracted, but during them, they became greatly dilated, and the eye-balls were drawn upwards and inwards. In twelve hours she had eight fits, and as each lasted about ten minutes, there was an average interval of about an hour and twenty minutes between them.

The head was shaved, and a large blister placed over the occiput and down the back of the neck, and a quarter of a grain of extract of elaterium was administered every three hours. She was allowed small quantities of beef-tea and a little wine.

She was soon freely purged by the elaterium, and the fits ceased on the evening of the 31st. On the 1st of June she had quite recovered her consciousness, and from this time she gradually improved, still under the influence of elaterium. The quantity of urine increased considerably, and at the same time free watery discharges were passed five or six times a day from the bowels. The dropsy disappeared, and the heart's action improved in force. On the 29th of June, the elaterium was discontinued altogether.

On the 3rd of July the urine diminished in quantity from a quart and six ounces to a pint and a half, and a slight fit occurred in the night; the elaterium was resumed on the 4th. From

this time the urine increased rapidly, and the quantity of albumen in it became much diminished. Although weak, she left the hospital on the 20th, being anxious to return home.

This patient continued pretty well until the beginning of October. On the evening of the 6th of that month, she was brought to the hospital quite insensible, and suffering from a succession of epileptic fits, just of the same nature, and as violent as before, but with such rapidity, that the intervals between them did not allow of anything being given, either food or medicine. They continued throughout the greatest part of the night, and she died early the next morning, not a gleam of consciousness having shown itself.

The examination of the body took place on the following day.

The kidneys were small, contracted, and seemed much wasted at the expense of the cortical substance, the cones in many instances reaching almost the very surface of the organ; in some of the cones there were opaque streaks of deposit of lithate of soda, taking the direction of the tubes, and probably occupying the canals of some of them. There was no healthy epithelium in any part of the cortical substance; in some situations the cells were filled with oil, in other places they were opaque. The walls of the minute arteries were thickened and hypertrophied. Each kidney weighed only three ounces and a half.

The heart was very large—it weighed nineteen ounces when deprived of its contained coagula. All its cavities were dilated and hypertrophied. The left ventricle was especially thick and large. The aortic and mitral valves were slightly opaque by interstitial deposit, and the chordæ tendineæ seemed thick and shrunk. In the dilatation of the left vetricle and the shortened chordæ tendineæ, there was ample explanation of the imperfect action of the mitral valves. There was no obstruction in the large arteries to explain the great hypertrophy of the left ventricle; that lesion seemed most probably to be due to the thickened state of the capillary arteries of the kidneys, and the consequently impeded circulation through those organs.

The brain was carefully sliced, and to all appearance it was perfectly healthy; its substance generally was very firm; it was everywhere paler than usual; there was no morbid condition of the membranes. The specific gravity of the grey and white matter was carefully ascertained by Dr. Conway Evans, by the ordinary method for determining the density of a body heavier than distilled water, and insoluble in that fluid. The density of the grey matter of the *convolutions* was found to be 1032·71, and that of the white matter of the *centrum ovale* 1037·17, water being considered as 1000. On microscopic examination, the minute cerebral blood-vessels pre-

sented no indication of fatty or earthy degeneration.

The brain was subjected to chemical analysis, with the view of detecting the presence of urea. This was performed in the following manner:— About three-fourths of the whole brain (including more or less of each of its segments) was cut up into small pieces, and treated with four successive portions of boiling distilled water, each portion, consisting of about ten ounces, being allowed to stand six or eight hours before the next was added. The brain, while thus macerating, was frequently stirred and mashed about with a glass rod. The washings, after being poured off, were mixed together and filtered. The filtered aqueous extract so obtained was now evaporated to dryness over a water-bath, and the dry residue, after being powdered, was again treated with four successive portions of boiling distilled water, observing the same precautions as before. The washings, after being mixed together, as before, were filtered, and the clear solution evaporated to dryness over a water-bath, and after being thoroughly dried in a hot-water oven, the residue obtained in this manner was finely powdered and the powder boiled in five successive portions of ether. The ethereal extract so obtained was evaporated to dryness at a low temperature, and then treated with a little tepid water and allowed to get quite cold. It was then filtered through paper

previously moistened with water, and the clear solution again evaporated to dryness at a low temperature, when a small quantity of the extract procured in this way (which would contain all the urea present in the brain operated upon) was placed on a glass slide, treated with a drop of strong nitric acid, covered with a bit of thin glass, and allowed to stand a little time; and then, examined under the microscope, a few crystals were seen, having all the characters of those of nitrate of urea.*

* In this, and two other cases of renal epileptic coma, I have sought, in vain, for evidence of the presence of carbonate of ammonia in the expired air and in the blood, as suggested by Frerichs. (*Die Brightsche Nierenkrankheit.*)

LECTURE XVII.

On a Case of Syphilitic Disease of the Dura Mater, and Periostitis.

I SHALL make to-day, Gentlemen, some remarks upon the case of a patient who, I find, has left the hospital. You have had, however, quite enough opportunity to make yourselves acquainted with all the particulars of it, as he has been some time under our observation, and I have frequently at the bedside pointed out the leading features of his malady. A clinical lecture delivered in the theatre, apart from the patient, has this great advantage, that we can discuss freely all the points of a case, many of which cannot be equally well commented on at the bedside, and some *ought* not to be referred to in the presence of the patient.

This is a case of peculiar importance and interest. It is an example of the way in which secondary syphilis affects the periosteum of different, and even important parts, producing serious, and possibly fatal, disturbance in their functions. Here you have first primary and then secondary syphilis; then periostitis, first of external parts, and subsequently affecting the dura mater; then symptoms of brain disease, both local and distant;

and, lastly, when the nature of the disease was detected, rapid and perfect relief.

Case LXXIII. The patient was James Beglin, a compositor, æt. 34 (vol. xxvi.), admitted June 6th, 1851: the history of the case illustrates very well the ordinary course of many an ill-managed syphilitic case; it is this—that fourteen or fifteen years ago he contracted syphilis, and had a chancre and a bubo: for these he took, without medical advice, a large quantity of mercury, which salivated him freely, he took as much as 210 grains, or ʒiiiss, of blue pill, besides the external application of a great quantity of mercurial ointment. The consequence was, his gums became spongy, his teeth loose, his glands swollen.

After the chancre and bubo had healed, he continued well for seven months, and then an eruption came out over his legs, arms, back, and face, &c. For this he was treated with solution of antimony and nitric acid (he thinks), and in a short time cured. Soon after the disappearance of the eruption, symptoms of iritis came on. His description of the affection of his eyes is a tolerably exact and clear account of an attack of iritis. He says he first perceived a haziness before his eyes; they were very much inflamed, vision was much impaired, and he had belladonna applied. This makes it probable that some adhesion had taken place, or threatened to take place, between the

margin of the iris and the anterior surface of the crystalline lens.

From all these evils he seems to have escaped pretty well, and he got married. He appears to have had no return of any unfavourable symptom till five years ago, when he applied as an out-patient to one of the hospitals in the metropolis for contraction and rigidity of the flexor muscles of the right forearm, and numbness in the same region. The nature of this symptom was not recognised; the case was looked upon as surgical, and the affection as local. The surgeon into whose hands he fell first applied leeches, then warm fomentations day and night; then, thinking there was some deep-seated abscess at the root of it, he made several free incisions, but all with no effect: so great was the contraction that it was in contemplation to divide the tendons. Fortunately, however, this was not done, and the patient left the hospital, after having been in it twelve weeks, just as bad as when he entered it. The contraction seems to have been confined to the flexor muscles of the forearm: the elbow-joint was moved with perfect freedom, the wrist slightly, but the fingers were perfectly stiff. After leaving that hospital, he went from one to another without deriving any benefit. At last, a singular accident happened to him: he met a friend, to whom he related his symptoms, and who told him that he had suffered from an exactly similar attack,

and that he had been cured by a certain medicine, of which he would give him the prescription, and which he had no doubt would cure him likewise. He accordingly took it in the prescribed dose, and it *did* cure him. In three weeks he was perfectly well, and not only had the contraction subsided, but the sensibility was restored, and from feeling very ill, and having no appetite, he became quite well and strong, and his appetite recovered. The medicine turned out to be iodide of potassium.

From that time he continued quite well, till six months ago, when he was thrown out of work, and got into that mentally and physically low state which is peculiarly favourable to the development of any disease, the seeds of which may have previously existed in the constitution. In consequence of this he went to Ireland, where he had some friends, and there, in Dublin, about four weeks ago, he had a fit, accompanied and preceded by severe pain in the head. He became alarmed, and returned to London, and shortly afterwards applied to this hospital as an out-patient, where the syphilitic nature of his disease was at once recognised by Mr. Bowman. He intended to continue as an out-patient, but having had a third fit the day after, determined to become an in-patient at once, and was admitted on Saturday, May 5th.

On his admission he appeared in a very low condition: his face was pallid, and he spoke tre-

mulously: he complained principally of pain in his head, situated in the region of the left parietal bone, and also of great pain and tenderness over the head of the fibula of the left leg. The pain in the parietal region was not increased by pressure, but the region of the head of the fibula was much swollen, and extremely tender to the touch. The pain both in the head and the fibula was severe, and became so much aggravated at night, as to deprive him of his rest,—a symptom bearing distinctly on the diagnosis. The situation of the pain in the leg was marked by a swelling of considerable size, which appeared to implicate not only the periosteum, but the ligaments of the joint and the fascia. At the situation of the pain in the head, there was nothing to be seen or felt; there was no evidence of inflammation affecting the pericranium; but the pain was very defined, both in character and in situation; it was fixed with variable severity in the parietal region, and never left it nor extended beyond it.

In addition to these symptoms, there was a very remarkable state of the *right* forearm—the side, you will observe, opposite to that on which the pain in the head existed. The muscles of the forearm were all in a rigid state, and the fingers forcibly bent into the palm of the hand, the flexor tendons being rendered quite tense by the firm contraction of the muscles. Moreover, there was a good deal of numbness of the forearm, and the

rigid state of the muscles both impeded their action and weakened them. There was, in fact, imperfect paralysis both of sensation and motion.

So far, then, we have all the history of the case which can bear upon or be necessary for the diagnosis. I regret that we omitted to make inquiry into one point very interesting in reference to general pathology. He had been married within the last few years; and we ought to have inquired whether his wife had become pregnant, and whether she had gone her full time, and borne healthy or diseased children. That a man still tainted with syphilis will beget syphilitic children without exciting primary symptoms in the woman, is now well known; and the tendency which women impregnated by such men have to miscarriages is also a fact of great interest, especially with reference to the laws of the humoral pathology.

Now the existence of periostitis was quite evident, from the large swelling over the head of the fibula; and the history of the case, the aggravation of the symptoms at night, the influence of iodide of potassium, made this part of the diagnosis easy and certain; but what was the connexion between the periostitis and the condition of the arm? Was it dependent on some local change affecting the periosteum of the bones of the forearm, and the tendons of the muscles, and perhaps also the nerves; or was it due to some

morbid state of a distant part—the brain, for instance? I thought for a moment that the contraction and partial paralysis of the forearm might have been dependent on irritation from periostitis affecting the part itself; but very little reflection convinced me that an affection so extensive, and of such long duration, was not likely to result from a local cause, nor could I call to mind any case in which such symptoms resulted from a cause of that kind. On looking further into the case, I came to the conclusion, that the affection of the arm was caused by syphilitic inflammation of the dura mater—the internal periosteum of the cranial bones—in short, by an intracranial node, which caused pressure on the surface of the cerebral hemisphere, and gave rise to paralysis, with irritation and consequent muscular rigidity.

This diagnosis was founded on the following points, which our investigation of the case enabled us to make out. First, there was paralysis not only of motion, but also of sensation: this denoted that the paralytic state was due to an affection of the *nerves* rather than of the muscles. Secondly, the absence of all evidence of node, or other periosteal affection of the bones of the forearm, rendered a local cause very unlikely. Thirdly, the general history of the case distinctly denoted an affection of a syphilitic nature. And lastly, the fixed and constant posi-

tion of the pain in the head, situated as it was on the side opposite to that on which the paralytic affection existed, indicated mischief in that situation. The occurrence of epileptic fits after the pain in the head and the affection of the arm had existed for some time, evidently associated that pain with cerebral disturbance.

The train of phenomena in this case was as follows. There was first syphilitic inflammation of the dura mater: this was slow and insidious in its progress, and did not at first cause any great pain: it then produced a certain amount of node; and this, by its pressure upon the surface of the hemisphere, caused irritation and paralysis, accompanied by spasm and rigidity, or an inflammatory state of the arachnoid and pia mater was induced, corresponding to the position of the inflamed dura mater, and exciting irritation of the surface of the brain; and, as the pain in the head increased, the paralysis both of sensation and of motion increased. The paralysis, imperfect as it was, and accompanied by rigidity, was just such as would arise from pressure or irritation on the *surface* of the brain.

I think, then, there can be no doubt that in this case we had syphilitic inflammation of the dura mater, exactly analogous to that affecting the periosteum of the fibula. Idiopathic inflammation of the dura mater is extremely rare: indeed, taking the word *idiopathic* in its strict

sense, as implying the absence either of constitutional taint or of a poison introduced from without, I should say it never occurred. Besides local injuries, scrofula and syphilis are the usual causes of inflammation of the dura mater: of the scrofulous form we sometimes meet with an example in that produced by otitis spreading from the ear to the cranium, the primary disease being almost always scrofulous in its origin. Indeed, the further we advance in our knowledge of pathology, the more shall we find it necessary to give up ideas of idiopathic inflammations, and the more certainly shall we be able to trace inflammatory affections to some defect in the excretory functions, or to the introduction of some morbific material.

If anything further could be wanted to support this diagnosis, it is supplied by the perfect success of the treatment which was based upon it. From the history Beglin gave us, it appeared that his symptoms on a former occasion yielded to the influence of iodine. On his admission into the hospital he was put upon three grains of iodide of potassium three times a day. The effect was marvellous: almost as soon as it was given to him the symptoms began rapidly to give way, as the following daily report shows:—On the 9th—three days after admission—the report is that the patient seems decidedly better, has an appetite, pain easier, looks better, sleeps well.

On the 12th.—Better in every way; is able to use his arm; pain in the head gone, in the leg less; swelling diminished.

17th.—Better in every way; pains in the head and leg quite gone; sleeps, eats, and drinks well.

19th.—Quite well in every respect.

Now, if we had failed in our diagnosis, we might have kept this man for weeks in the hospital without doing him any good, and we might perhaps have sent him away just as he entered, instead of dismissing him cured of his malady, and in a state fit to resume his ordinary avocations. Nothing can give a clearer idea of the importance of a correct diagnosis than the history of a case like this. If the disease had not been arrested, it is probable that more lymph would have been poured out upon the dura mater—the pressure would have been augmented—the fits would have recurred; and the epilepsy, instead of being transitory, would have become confirmed: in fact, it is impossible to say where the effects of the disease would have stopped: whereas, by a careful investigation of the case, a due appreciation of the indications furnished by its history, and the administration of a small quantity of a medicine which may be always safely given within moderate limits, the man is restored to perfect health. If any one were inclined to attach but little importance to correct diagnosis, the instance of such a case as this

is all that is necessary to prove the danger and absurdity of such views. It shows that the great step towards cure is the right understanding of the nature of the disease with which we have to grapple.

Now I ask—What has been the exact state of the dura mater in this case? I think the best way to answer this question will be to give you an account of the history and post-mortem examination of a case that occurred in my hospital-practice some years ago. These cases are generally capable of cure, if detected early; but if allowed to go on too long, new products are formed which permanently damage the brain, and are not susceptible of cure. The case to which I allude is as follows :—

Case LXXIV. A woman, æt. 31 (vol. ii., p. 37), was admitted into the hospital some years ago, suffering from the following symptoms :—great loss of memory, fits, imperfect palsy of the left side, and pain in the right parietal region. She was a married woman and had lost her husband, and she attributes her illness to grief from this cause. About this time, which was between five and six years before her admission, the pain in her head began. She led an irregular life, was intemperate, and, being of easy virtue, contracted syphilis, and had chancre, sore throat, papular eruption, and nodes on the tibiæ. Two months

before her admission she had her first fit: it came on shortly after she had eaten a hearty supper. She became insensible, was much convulsed, especially on the left side, bit her tongue, and after the convulsions had ceased she remained in a drowsy state for twelve hours. After this the pain in her head increased considerably, and was more fixed and defined: it was referred to the right parietal region. The fits likewise recurred frequently; they were always preceded by a sense of painful numbness in the left hand and foot. The *right* pupil was occasionally more dilated than the left, and the right upper eyelid hung lower than the left, as if the third pair of nerves on the right side were slightly affected.

Despite of active treatment, bleeding, local and general, the free exhibition of mercury, and the long issue in the scalp, this woman became more and more comatose, and had a severe fit from which she never rallied. On examination we found the dura mater firmly adherent to the right parietal bone, and also to the visceral layer of the arachnoid, for an oval space two inches in its long, and an inch and a half in its short diameter. At this part the dura mater was three or four times its natural thickness. Between the layers of the arachnoid, there were two large masses of a yellow colour, like concrete pus, opposite to which were corresponding de-

pressions or concavities on the surface of the cerebral hemispheres. At these points the cerebral substance was slightly softened, and redder than was natural. At the corresponding point on the opposite side of the skull a similar morbid alteration in the dura mater and the arachnoid was commencing. There was no disease in any other part of the brain.

Now this is a good typical case, illustrative of the way in which syphilis may affect the parts within the cranium, and the extent, when not early arrested, to which it may proceed. The dura mater, like any external periosteum, becomes affected by the syphilitic poison; then follows arachnitis; and lastly, cerebral compression, and perhaps red softening. It is not likely that our patient, Beglin, had such extensive disease as we found with this woman, otherwise he would not have recovered from the paralytic affection so quickly. But I cannot doubt that changes of this kind would have taken place within his cranium if the progress of the disease had not been arrested. If the woman, whose case I have just related, had come into the hospital three or four months before the time she did, and had been put under a course of iodide of potassium, or other suitable remedies, it seems certain that she would have completely recovered.

Let me give you another example of a case of this kind, in which the disease had not

gone so far as to be beyond the power of remedies.

Case LXXV. The patient was a young man, who followed the business of a chemist and druggist. There was a distinct history of syphilis. He had experienced a good deal of pain in the right parietal region, which had been treated as neuralgia. From this treatment no benefit resulted, and shortly afterwards he began to suffer from imperfect paralysis of the left arm and leg; and I noticed decided thickness of speech, his manner became dull, and his spirits much depressed. The paralysis then became general on the left side, affecting his face, and the scalp became tender over the painful region. I determined to put him under treatment calculated to relieve inflammation of the meninges, and accordingly gave him calomel and opium, and applied tartar emetic ointment to his head. In ten days his paralysis had vanished, and all his other symptoms had left him.

I may refer briefly to another case, which you have frequent opportunities of witnessing, as he is often an inmate of the hospital: his name is Coulson, and he affords a good example of very complete paralysis of the fifth nerve of the left side. I have no doubt that syphilis is at the root of the disease in this instance too. The man is subject to epileptic fits, the immediate exciting

cause of which is intemperance. I think the exact diseased condition in this case is syphilitic inflammation of the dura mater in the vicinity of the fifth nerve and the Casserian ganglion; that lymph has been poured out, which presses upon the ganglion, and, including both the sensitive and motor portions of the nerve, suspends alike their respective functions.*

There are one or two more points touching cases of this class, with which I shall conclude what I have to say to you to-day.

And, first, as to the cause of the epileptic fits which occurred in three out of the four cases, and which I can scarcely doubt must have occurred in the fourth case had it been suffered to proceed much further. The occurrence of epileptic fits in such cases as these appears to me to denote that the brain itself is involved in the morbid action, either by extension of irritation from the diseased parts, or by the direct influence of the syphilitic poison; but in all the cases the simple existence of the diseased state of the dura mater did not seem sufficient to cause the fits. The morbid process had been of long continuance in the dura

* This patient died many months after this lecture was given, and the arachnoid sac was found completely obliterated by old cellular adhesion all around the fifth nerve. The trunk of the nerve was wasted, and appeared under the microscope to consist solely of wavy fibres of white fibrous tissue.

mater before the fits began. They seem to have been brought about by causes operating on the whole system—upon the blood—upon general nutrition—and upon that of the brain in particular, which must have already suffered a good deal by the neighbouring disease of the dura mater.

In some cases of meningeal disease, especially, I think, when the pia mater has been previously affected, we meet with fits of a very remarkable character: I have called them *epileptiform*. One arm, or both arm and leg on one side, become seized with convulsive movements, quite of the clonic or epileptic kind. These come in paroxysms; the paroxym lasts a variable time, and then subsides, leaving more or less general exhaustion and disposition to sleep; but consciousness is not impaired. Yet there can be no doubt that such fits may pass into the true epileptic fit; for it is not rare to see a very complete epileptic fit commence with some local derangement of sensation or motion, or both.

Secondly, the extraordinary influence of iodide of potassium in controlling syphilitic periostitis is highly deserving of your attention. We hear a great deal about specifics, and many medicines have the name with very slender claims to it; but if there is anything, in addition to quinine, which deserves the name of a specific, it is the iodide of potassium, for syphilitic periostitis. If

you have a *pure* case, it acts like a charm, so that the treatment materially aids the diagnosis; for we may fairly set it down, that if the symptoms yield completely and at once to the influence of iodide of potassium, there is strong reason to suspect that the disease is probably syphilitic.*

But, although iodide of potassium unquestionably exerts a wonderful influence over these syphilitic periosteal affections, nothing is more certain than that its effect is far from being permanent. It is very common to meet with cases of periostitis which at first yield readily to the influence of iodide of potassium, and the patient continues well for a time, when all the old symptoms return. It was thus in Beglin's case: some time before his admission into the hospital he took iodide of potassium, and the symptoms yielded; but they afterwards returned, and were again removed under the influence of the same remedy. I think it not at all improbable that

* In making this statement I believe I do not go too far; although it is not absolutely true that iodide of potassium is only useful in syphilitic periostitis. I have seen cases of periosteal rheumatism and gout affecting the fibrous tissue greatly benefited by it; and I have seen it do marked good in periostitis after fever. At the end of this lecture will be found a case very similar to that of Beglin, of which the apparent origin was an injury, and yet the disease yielded only to the iodide of potassium.

he may again have a return of the symptoms, and may once more come into our hands.*

The knowledge of these clinical facts teaches us that we must not speedily abandon the use of the iodide, or of iodine in some other form, in cases of this description. In the present state of our knowledge we can scarcely determine whether the iodine acts by eliminating the syphilitic poison, or as an antidote. Possibly it may act in both ways; it may at once promote the action of emunctories, and so increase the amount of matters excreted from the blood, and unite with the syphilitic poison, forming an innocuous compound, of which, however, the iodine element disappears more quickly than the syphilitic, leaving, after a time, the syphilitic in undisputed sway in the system.

In such cases we must trust to the repeated use of iodine as one element of cure, care being taken to watch the constitution of the patient during its administration. And we may aid the influence of the iodine by the occasional use of mercury, either at the same time with the mercury, or, as I prefer it, alternately,—that is, giving first a short course of mercury, then of iodine, then of mercury, and then omitting both, and using only tonic means, both medicinal and hygienic, resuming, if occasion should demand,

* Vide the sequel of the case, pp. 398, et sqq.

the mercurial and iodine treatment. And you will also find great benefit from the prolonged use of well-made decoction of sarsaparilla, or of cod's liver oil, or of both.

But you must never lose sight of the fact, that *time* is an important element of cure in these cases; and, therefore, we must be careful not to weaken the powers of our patients by our treatment; lest, by so doing, we should not only retard their favourable progress, but give a stronger hold to the poison. We have seen, in the history of Beglin, how a state of enfeebled nutrition from bad living clearly favoured the development of the serious symptoms. It will be our duty, then, to encourage our patients to expect a favourable issue; to impress upon them the necessity of a steady perseverance in a general plan of treatment, and to point out the dangers of swerving from the hygienic or dietetic rules laid down for them.*

I am fortunately enabled to follow up the further history of this patient Beglin. It was on the 22nd of May, 1849, when the preceding lecture was given upon his case. He had then just left the hospital. From that time till June, 1851, he continued pretty well; but he would call every now and then at the hospital, when

* The lecture, as originally given, ended here.

suffering from pain in the head, which I suspect was often aggravated by intemperate habits and unhappy domestic relations.

Early on the 9th of June, 1851, he was brought into the hospital in violent convulsions, which were confined to the right side, the left being moved only by the action of the right. These lasted till 6 P.M., and in the night he had several short fits. On the 10th there was still some slight convulsive paroxysms, each lasting a minute. He had quite recovered his consciousness, but not his speech, and pointed to the left side of his head as the seat of his pain, and protruded the tongue when told to do so. On the 11th he had a very violent fit, in which the body was bent over to the right side, with powerful, clonic spasms. The tongue was bitten on the right side. The urine was ascertained to be quite normal. Towards evening the fits subsided, and he was able to speak a little.

He had a blister applied to the shaven scalp, and two grains of calomel were given every four hours, mercurial ointment and savin ointment were applied to the blistered surface, and he was purged. The fits continued till the evening of the 16th, when he showed evident signs of salivation.

On that day they had been very severe, still affecting the right side. They ceased during the night, and at 7 A.M., on the morning of the 17th,

he had one fit, and another at 11 o'clock, A.M. These were the last. Then followed a state of noisy delirium, which gradually subsided, under the use of opium and stimulants, in a week. He now complained chiefly of muscular pains, weakness and numbness of the right arm and leg, and of pain in the head on the left side. Five grains of the iodide of potassium were prescribed thrice a day, and counter-irritation of the scalp was kept up opposite the seat of pain. From this time he improved steadily, and left the hospital in his ordinary state of health on the 18th of July.

From this time till the end of the year 1852, Beglin applied at the hospital occasionally, suffering from pains in the head, and in the right arm and leg. I do not know whether he had any fits during that time. There was no doubt that his habits were careless and rather intemperate, and such as would keep up the epileptic tendency. On the 7th of December, 1852, he was seized with severe pain in the left parietal region, having been out of sorts and unable to attend to business for some days previously. During this time he often had recourse to brandy-and-water to rouse him. The pain increased very much, and he suffered from frequent paroxysms of it during the whole of that day, Monday, and on the following day, Tuesday. On the Wednesday morning he was seized with a succession of fits, affecting

chiefly the right side; after the eighth of which he sank rapidly. On this occasion he had not been brought to the hospital, nor had any iodide of potassium been administered. Indeed, from the description given by his sister, it seemed that the fits were so violent, and succeeded each other with so much rapidity, that it was not likely that any ordinary remedy would have controlled them.

This poor man, knowing the interest we took in his case, left instructions that I should be informed of his decease, and permitted to examine his body. This, with the assistance of my friend, Dr. Hyde Salter, was accordingly done.

The body was in a state of good nutrition and was tolerably fat.

On sawing through the skull, the bone in the right temporal region was found much thickened. A small osseous spiculum projected from the inner surface of the left temporal bone.

The dura mater was easily separated from the calvaria. Opposite the thickened bone it was also slightly thickened. It adhered very closely to the arachnoid and pia mater on each side of the falx, and in its whole length. Here the Pacchionian bodies were very large, and adhered so intimately, that in removing the dura mater the pia mater came with it. The arachnoid was quite opaque in this situation. At the posterior and upper part of the left parietal region, and at

about an inch from the longitudinal fissure, the dura mater was adherent to the subjacent membranes for about the space of a square inch, and so firmly, that in taking it off, it brought away not only the pia mater, but the cortical substance to a considerable extent.

The grey matter of the convolutions was darker than is natural; this was especially remarkable in the convolutions corresponding to the adherent membranes on the left side, as above described. Here the brain substance seemed slightly hardened. The convolutions generally had a shrunk appearance, and the sulci were wide, and the subarachnoid fluid existed in considerable quantity. On the surface of each hemisphere, just above the upward termination of the fissure of Sylvius, there was a depression, as if one or more convolutions had completely wasted away. The pia mater occupied the depression, which corresponded in size and position precisely on both sides. Unfortunately it was found impossible to examine the brain microscopically.

All the other viscera were healthy.

The amount of recent disease here was out of proportion to the severity of the symptoms during the last day or two of life; and the evidence of old lesion, on the left side of the brain, was scarcely as much as one might have expected from the violent convulsions and severe pain, were it not that we knew that the patient had been sub-

jected to very active treatment by mercury, iodide of potassium, and counter-irritation a year and a half before. And knowing how completely considerable periosteal thickenings on the shins and elsewhere disappear under similar remedies, it was satisfactory to find no greater marks of disease remaining within the cranium.

It can scarcely be doubted, I think, that had this man been in easy circumstances, and of temperate habits, he might have been completely cured, and that the epileptic attacks would have ceased, or assumed a much milder form.

I am indebted to my friend, Dr. Kennion, of Harrogate, for the particulars of a very interesting case, presenting many points of similarity to that of Beglin, and cured by the exhibition of iodide of potassium, but differing in the absence of any syphilitic taint.

Case LXXVI. An officer in the Queen's service, while on duty in one of the colonies in September, 1851, received a violent blow from a pistol on the forehead, which stunned him for a short time, but the immediate effects soon passed off. Two days afterwards he was seized with a violent pain in the right temporal bone, which was exceedingly tender to the touch, but was not swollen or red. Being actively engaged in service, he used no remedies; he fell off rapidly in flesh and strength. Four weeks after this he fell

from his horse in an epileptic fit, but without suffering any injury by the fall. He was now sent home, and arrived in England in January, 1852. From that time until June he suffered agonies from the pain; he had various returns of the epileptic seizures, and continued to lose flesh. In March a swelling was perceived for the first time in the right temporal region.

Mercury, purging, leeches, blisters, had all been freely employed in this case without the slightest beneficial effect. At length it was determined in consultation to suspend all other treatment, and to give large doses of iodide of potassium. At first he took five grains three times a day, and the dose was gradually increased to fifteen grains without producing any unpleasant physiological effect. Within a fortnight after the commencement of the iodine, the pain diminished in severity, and at the end of two months it entirely subsided. He had no return of the fits since August, at the date of Dr. Kennion's communication to me (November 22, 1852).

The influence of the iodide of potassium in these cases is of extreme interest. Periostitis, or, which is the same thing, inflammation of the dura mater, may be excited in strumous, rheumatic, or gouty states of constitution, and more probably in persons tainted with syphilis. The facts, both of clinical history and of treatment, bear upon the pathology and treatment of those cases of epilepsy,

which follow the syphilitic taint, and point to the iodide, and to mercury and sarsaparilla, as the remedies which ought never to be lost sight of in such cases.*

* I have lately seen with Mr. W. J. Jones, a middle-aged man, of irregular habits, who had primary syphilis some years ago. Shortly before my visit a considerable node formed on the right parietal bone. He suffered very much from pain in the head, and from giddiness, and he had several epileptic fits, which left him very much exhausted. After the first fit he had complete paralysis of the left arm, which lasted only half an hour. I prescribed iodide of potassium in large doses, and he speedily recovered.

In connexion with the subject of this lecture, interesting cases and valuable remarks will be found in Sir Everard Home's paper entitled, "Cases and Observations which show that inflammation is sometimes communicated from the dura mater to the pericranium" (Transactions of a Society, &c., vol. iii., p. 122); in the same volume, there is a striking case by Mr. Wilson, p. 115; reference may also be made to a paper by Sir Philip Crampton, on Periostitis, in the Dublin Hospital Reports, vol. i.; and to a valuable Lecture in Dr. Graves' Clinical Lectures, vol. ii.; and also an interesting paper by Dr. Reade, of Belfast, in the Dublin Medical Journal, vol. xiii., new series; and a case by Dr. McDowel, in the Dublin Hospital Gazette for April 15, 1854.

LECTURE XVIII.

On a Case of Acute Idiopathic Trismus.

WITHIN the last day or two, as you are aware, a patient has died within the walls of this hospital of idiopathic tetanus, or, more properly speaking, of that form of it which, from the muscles of the jaws being chiefly affected, is called trismus (τριζω, strido); and, as this is comparatively a rare disease, and, in the majority of cases, a fatal one, I am anxious not to let an instance of it pass without making it a source of instruction to you, by giving you some account of its clinical history and pathology.

CASE LXXVII. The patient, Henry Franklin, æt. 40, was admitted into the Sutherland ward on Tuesday, November 6th, 1849. The following history of him was obtained:—On Sunday, October 28th, he awoke with a swollen face, and some feeling of stiffness about the jaws; he attributed this to having caught cold, by sleeping with a window open. The feeling of stiffness increased, but he was able to swallow until Sunday, the 4th of November, seven days after the first accession of the symptoms; but since that time

he has been unable to perform the act of deglutition. On his admission (Tuesday, 6th) we found him with the face very much swollen in the region of the parotids, and with considerable fœtor of his breath; and it was ascertained that he had been taking some pills which contained a small quantity of mercury: it is therefore possible that he may have been one of those persons who are affected by a small quantity of mercury, and that he was in a state of salivation.

We found also great rigidity of the masseter muscles, so as to prevent him from opening his mouth, and from either speaking or swallowing. His teeth could, at first, be separated just so far as to slip in a card, but no farther. The attempt to separate the jaws caused great pain, as is generally the case, if tension is applied to a muscle in a state of contraction, as in ordinary cramp, and in the treatment of club-foot. It appeared that the muscles of deglutition were also affected, as he stated that the attempt to swallow his saliva nearly choked him.

Upon further inquiry, we learned that his previous life had been temperate and sober; that he had had syphilis and secondary eruption, but no sore-throat or nodes on his tibia or elsewhere; that he had never been subject to fits of any sort, but that he had had brain-fever. This was the first day we saw him, and my apprehensions were excited as to the nature of the case: I feared it

would turn out what it afterwards proved. But this supposition was opposed by the absence of any manifest cause for tetanus. Excepting the exposure to cold, the slight salivation, and a small abrasion on the nose, occasioned by a fall that occurred the day before the accession of his first symptoms, there was no cause to which tetanus could possibly be assigned. The wound on the nose was very slight, and may be dismissed from the consideration at once. Cold is not an unfrequent cause of tetanus in tropical climates, where the alternations of temperature are great; but it is a rare cause in this climate. I have witnessed one such case, in which the tetanic symptoms followed the exposure of the patient to the open air during the whole of a very cold night. It is very possible that the exposure to the open window, all night, might have excited the disease in the present instance. Might the salivation have caused the tetanus? There are on record some cases of tetanus produced by salivation. But it does not appear from the history of this case, that the salivation was distinctly antecedent to the tetanic symptoms.

There was a certain amount of obscurity as to the origin of the disease, and I was not quite prepared to act at once upon the notion that it was tetanus; I therefore adopted a treatment less decided than I should otherwise have pursued. I ordered him fomentations to the jaws and

throat, an enema of starch and opium at night, and beef-tea enemata throughout the day: in fact, my treatment was very much of the expectant kind. I determined to see what the next day would produce, and contented myself with dealing with the existing symptoms.

The next day we found the symptoms of spasm still the same. The jaw was firmly clenched, and attempts to open the mouth produced great pain. He was still unable to swallow his saliva, which frothed out of his mouth at each expiration. The secretion was certainly unduly abundant. All this time he was perfectly sensible, clear, and composed. He was not at all excited in his manner, and, although he could not speak, he understood all that was said, and wrote down on paper coherent and intelligent answers to all questions that were put to him.

Here, then, we had no symptom but the local affection of trismus, which might have been the effect of the local application of cold; but my fears were that it was but the early stage of a genuine case of lock-jaw, and I stated my opinion to that effect. I therefore determined to try tobacco fomentation, which I have frequently seen used, and with advantage, care being taken to uphold the strength of the patient. Tobacco has this great disadvantage,—it is a very depressing agent, and therefore tends to throw the patient into that very condition which would favour the

fatal tendency of the disease. To avoid this, care should be taken to use the tobacco infusion weak, or the patient may be exhausted to a degree that may prove fatal.

At seven o'clock of the evening of this day, a symptom supervened which left no doubt as to the nature of the malady: it consisted in a violent paroxysm, exciting to active contraction, not only the muscles of the jaw, but also those of the neck, chest, and back, the last producing partial opisthotonos; the sterno-hyoid, sterno-thyroid, and sterno-mastoid muscles were thrown into a state of rigidity, and this condition implicated the muscles of respiration, and so far interfered with that function, that the patient felt as if he should be choked, and jumped out of bed to endeavour to get air. This choking sensation was not caused by any contraction of the glottis, but was due to the impeded action of the muscles of respiration: in fact, the man was kept in a constant state of imperfect respiration, or rather of forced expiration, the consequence of which was that the venous system became highly congested, a good illustration of the way in which convulsion may give rise to congestion. The pupils were very much contracted *during the paroxysm*,—a circumstance of great interest, as showing a peculiar state of the nervous centres, connected with the excitation of the spasm; for, as soon as the spasm went off, the contraction of the pupils went off

too; and, when the spasm was at its height, the pupils were contracted to pins' points. The pulse during the paroxysms rose to 120.

From this time the case put on a more severe form: not only the permanent contraction of the muscles and all the other conditions continued, but every now and then one of the paroxysms would come on, each succeeding one being more severe and longer than that which preceded it; leaving the patient greatly exhausted, and exciting in our minds the fear that he might be asphyxiated and die in one of them. If, however, there had been reason for such a fear, there was nothing to be done to obviate it; for, even had we performed tracheotomy, it would have availed little, for we should still have left the rigidity of the respiratory muscles unrelieved, which alone would doubtless have sufficed to produce asphyxia. But, in truth, death by exhaustion was the thing to be most dreaded: what we had most to apprehend was that which actually did take place—namely, that the increased frequency and severity of the paroxysms would at length produce a fatal exhaustion, and that the patient would sink rapidly after one of them.

The indication for treatment was therefore to *support*; and since it was impossible to get the patient to swallow (the attempt being immediately followed by an exacerbation of the spasm), the only alternative was, in default of one entrance, to

try another. I gave him, therefore, a scruple of sulphate of quinine, in a small quantity of water, as an injection, continued the beef-tea enemata, and being anxious, if possible, to diminish the pain and spasm, I ordered the frequent inhalation of chloroform,—the plan being to give it, not in large quantities, but in small and often-repeated doses, with a large admixture of air, so as to produce a gradual and soothing effect. By this means we were able to obtain resolution of the spasmodic condition of the muscles; but to arrest the paroxysms, or diminish their strength when on him, the chloroform seemed almost powerless.

At about a quarter to twelve, A.M., on the 8th, the quinine enema was repeated: at this time he was lying in an unconscious state from the chloroform (under the influence of which he was constantly kept), with his muscles relaxed, and breathing tranquilly. About five minutes after twelve a violent spasm came on, the jaws knocked together with a distinctly audible click, the face became much distorted and livid, the eyes rolled upwards, and there was much frothing at the mouth, and great apparent difficulty of breathing, the sterno-mastoids violently contracted, and the head thrown forwards: in a minute or two this was succeeded by a well-marked opisthotonos, the body being much curved, and resting on the occiput and sacrum. In this state he died. His death may be said to have taken place by ex-

haustion, the immediate cause of which was the violent spasm.

There was nothing in the previous history of this patient to throw any light upon the case; he had been a sober man, had had syphilis and secondary eruption, and an illness which was called brain fever; there was nothing either in the man, or the circumstances in which he had been placed, that could be considered predisposing to tetanus. So it is in all the examples of this disease we meet with: there is no connexion between the previous history of the patient and the disease; the immediate exciting cause, and the detail of the symptoms, constitute the entire history of the case.

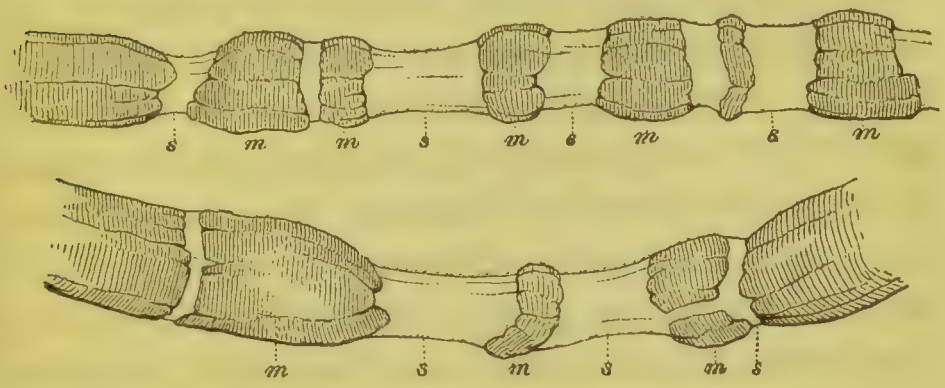
It fortunately happened that we were able to have a post-mortem examination, under very favourable circumstances. It was made only four hours after death, when, if there had been any morbid appearances, they would have been free from the fallacy of post-mortem change. There was, however, as I had anticipated, no morbid appearance which seemed to offer any explanation of the disease: the nervous centres, both brain and spinal cord, were perfectly healthy,—perhaps the brain contained a little more blood than usual,—a fact not to be wondered at when we consider the violence of the paroxysms: the grey matter of the brain was perfectly natural, and its demarcation peculiarly distinct: indeed, this remarkable

distinctness may have been morbid, and may have indicated an undue functional activity of these parts.

We examined the state of the masseter muscles, to ascertain if any change had taken place in their minute structure, in consequence of the prolonged spasm which they had undergone. The result has been confirmatory of certain doctrines first laid down by Mr. Bowman, in illustration of the way in which the contraction of muscle takes place. When a muscle is thrown into a state of contraction, that contraction does not affect the whole mass of the muscle, or the whole of any of its fibres, at the same moment, but certain points or nodes are contracted, while the intervening portions are stretched by the action of the contracted ones. Thus you have a succession of contracted points throughout the entire length of each fibre of the muscle. Here is a drawing made by Dr. Hyde Salter, of a magnified view of two fibres, taken from the masseter of the patient, showing the peculiar condition, which was first described by Mr. Bowman, as existing in the muscles of persons who have died of tetanus, and which, as you will see, is easily explained by the view of muscular contraction to which I have just referred. You must not imagine that all the fibres were so affected: these were the two most marked specimens among many hundreds that were examined, some possessing the appearance in a slight degree,

some not at all. Here you see the sarcolemma continuous throughout its entire length;

Two Fibres of the Masseter Muscle ruptured by the Tetanic Spasm.



m m m separated masses of sarcous particles; *s s s* sarcolemma.

the contained sarcous matter, however, is *not* continuous, but is broken up into isolated portions of various sizes, giving rise to a peculiar bulged condition of the sarcolemma, with collapsed constricted portions in the interval. Now how is this appearance produced? The explanation appears to me to be as follows: each of the separated masses was the centre of a contraction at the time, when the muscle was in a state of violent spasm; the force of the contraction exercised at these points was so great, that the tenacity of the intervening stretched portions was not sufficient to resist the divellent force: they gave way, and the particles thus isolated when in a state of contraction, having no antagonistic force to restore them when the contracting force had subsided, remain, as you see

them, in a state of permanent contraction, leaving intervals of considerable length between them.

These, gentlemen, are the main points to which I would call your attention in the history of this case; I shall now make a few observations of which this history is suggestive.

First, with regard to the name: what shall we call it? Two names are in frequent use for such affections—*tetanus* and *trismus*. I prefer *trismus*, as being more specific, localizing the affection, and showing its limitation to the muscles of the jaw. Tetanus is a generic name, and indicates the general existence of tonic spasm, without any indication as to its whereabouts. Tetanus can hardly be called a rare disease: trismus occurring in the adult *is a rare* disease. In the present case the affection was for eight days confined to the muscles closing the jaw; only within a day of his death did it extend to the muscles of the neck and back; and not till the very last stage did it so affect the muscles of the back as to produce opisthotonos: whereas, in ordinary tetanus, the extension of the spasm to all the muscles generally occurs early in the disease; opisthotonos may be a prominent symptom for three-fourths the duration of the attack; and, if you put your hand on the patient's arms or legs, you find the muscles hard and rigid; whereas, in the present case, the affection was limited, for nearly the whole time, to the muscles of the jaws. The only *essential*

difference, however, between the two diseases is in the extent of the nervous centres, and, therefore, of the muscles implicated.

Sir Benjamin Brodie and others have adopted an excellent practical distinction between *acute* and *chronic* tetanus—the one running a short course, with strongly-marked symptoms, and terminating always fatally; the other of longer duration and a milder form, and frequently recovered from. Traumatic tetanus is very apt to be acute; but tetanus, however caused, frequently puts on the chronic form. Some of these cases last twenty days or more; and, when prolonged in this way, there is great hope of ultimate recovery. If such a case—or still more, if two or three such—should happen to fall into the hands of some very zealous supporter of any particular practice—if he is a great bleeder, or a free giver of opium—the treatment is immediately set down as specific, and the recoveries are appealed to in proof of its infallibility, the real secret of the successful issue being, that the cases were chronic and not acute. Now in the case of our patient it cannot be said that any part of the treatment was likely to weaken the powers of life: possibly the means adopted for their maintenance and prolongation might have been more energetically plied; the quinine might have been earlier administered, and perhaps more support might have been given: still nothing was done that could by any possibi-

lity have accelerated the fatal termination of his disease. Yet we see him admitted into the hospital on the Tuesday with slight symptoms of the disease, and dead on the Thursday morning. And this is an essential part of the natural history of all these acute cases—speedy and fatal termination, death taking place by exhaustion.

The peculiar points in this case, that especially call for remark, are the violence of the spasm, and its restriction for so long a time to the jaws. As I said just now, genuine trismus is rare in adults. In newly-born infants, however, trismus is not rare, particularly in hot climates: in the West Indies it used to be very common: it goes by the name of *trismus nascentium;* and though now, from greater care in food, cleanliness, and ventilation, it is much less frequent than formerly, it still carries off a great number of children.

I have said that this case illustrates the mode of death by exhaustion: this is an important point, and one I particularly wish to impress upon you. Death does not take place by asphyxia, nor by any particular effect on any especial organ, but from general exhaustion, induced by the protracted and unwonted nervous and muscular excitement. This, I think, is not sufficiently attended to in the treatment both of this and of many other diseases.

The case also illustrates the little hope we have of obtaining, by post-mortem examination, any other than negative information as regards the

particular state of the nervous system: we can only draw our conclusions respecting the pathology of the disease from our knowledge of the physiology of the parts concerned. Now, reasoning on this principle, it may be laid down that the phenomena result from an exalted polarity of the centres supplying the parts affected. In the case of traumatic tetanus, the exaltation of the polar state commences in the afferent nerves of the seat of the wound: if the tetanus arise from cold, the exalted polarity commences in the nerves of common sensation distributed to the exposed part: from the periphery thus irritated the condition is propagated through the nerves to the centres, and the effects on the muscular system show to what portions of the nervous centres the exaltation of the polar force is communicated. This, however, does not afford an adequate explanation of the production of tetanus; for peripheral nerves, and even nervous centres, are often subjected to great irritation without giving rise to tetanus; and it is well known that it is impossible, even by severe mutilations, to produce tetanus in the lower animals: whereas a slight accidental injury (as when a horse picks up a nail) will often excite the disease in its worst form. It would seem that some peculiar state of the system—probably some peculiar condition of the blood—is a necessary precursor of this malady. Hence, no doubt, its greater frequency in warm

and unhealthy climates, in overcrowded and badly-ventilated military hospitals, and among ill-housed, ill-clad, and ill-fed infants.

That tetanus may be produced through the blood is shown by the results of the administration of strychnine, which imitate the tetanic symptoms in a very striking manner: so that you may at will develope the general phenomena of tetanus in an animal by giving him strychnine, or injecting it into his blood, but you cannot cause it by external injuries.*

* For further remarks on this subject, see my Lumleian Lectures for 1849, on the Pathology of Convulsive Diseases. The present Lecture was delivered in 1849, and printed in the London Medical Gazette for November 1849.

The diagnosis of the tetanoid condition produced by strychnine from the genuine tetanus, whether idiopathic or traumatic, has become a matter of medico-legal importance since the recent highly interesting trial of W. Palmer, of Rugeley, for the murder of J. P. Cook, through the agency of this powerful poison (June, 1856).

For the defence it was suggested that the tetanic convulsions under which Cook laboured for a short time on the Monday night, and more severely on the following Tuesday night, when he died, might have been due to idiopathic tetanus, or to traumatic tetanus, caused by sore throat. The tetanoid state from strychnine is only likely to be confounded with very acute tetanus, either traumatic or idiopathic. The existence of a wound would of course be some evidence in favour of tetanus; but, after all, the chief reliance must be placed on the symptoms. These are—

What was the exact portion of the centres affected in the present instance? The localization is pretty clear: it was evidently the upper part of the spinal cord, the medulla oblongata, and its upward prolongation in the cranium, especially in

1. As regards the tetanoid state from strychnine. *a.* The *rapid* supervention of tetanoid convulsions affecting chiefly and with most intensity the muscles of the trunk and spine, causing an active and violent opisthotonos rarely met with in true tetanus. *b.* A rigid and tetanoid state of the muscles of the lower extremities, with somewhat less intensity than those of the trunk: the limbs are extended, and the feet drawn powerfully inwards by the action of the tibiales postici muscles. The upper extremities are affected also, but in a less degree, and the hands are generally semiflexed. *c.* The trismus or closure of the jaws exists only imperfectly, and the *facies tetanica* very slightly, or not at all: swallowing is perfect, but the mode of deglutition seems to be peculiar, the patient snapping at the liquid offered, and gulping it down with an effort, in a manner very similar to that, in which hydrophobic patients swallow. *d.* The attacks of opisthotonos are very frequent, and seemingly exquisitely painful, and are ushered in by a cry more or less loud.

As regards tetanus—

a. The symptoms come on *gradually*, and the trismus is the earliest, the most prominent, and most important one. *b.* The facies tetanica is a very characteristic symptom. *c.* The attacks of opisthotonos are less frequent, less extensive, and less severe than in poisoning by strychnine. *d.* The extremities are the last parts affected, and suffer much less from the tonic spasms than other parts.

the neighbourhood of the implantation of the fifth nerve. Indeed, the supposition that the disease was caused by the salivation would afford a very plausible explanation of the localization of the morbid action in this latter region; for in ptyalism the seat of the peripheral irritation must necessarily be the sentient filaments of the fifth nerve, which would, of course, propagate the irritation to the centre, and excite a similar state of the motor nerves, thereby giving rise to the spasmodic condition of the muscles of mastication, which are supplied by the same nerve.

And we may ask, further, what is the nature of the morbid process set up in these centres? To this it may be confidently replied, as the result of repeated examinations, that it is not inflammation, nor anything allied thereto. And it may, with

e. Deglutition is slow and difficult, and sometimes impossible, owing to the spasmodic closure of the mouth.

In fine, trismus is the characteristic and primary feature of true tetanus; acute, violent and frequent attacks of opisthotonos, when not preceded by trismus, and when ushered in by a cry, consciousness being retained, are strongly indicative of poisoning by strychnine.

In the case of Cook, the diagnosis of strychnine poisoning was rendered certain by the occurrence of two distinct attacks of convulsions with opisthotonos, each preceded by the administration of suspicious pills, with an interval of *complete suspension* for twenty-four hours. Such a complete remission of symptoms never occurs in tetanus; nor could the convulsive phenomena be referred to epilepsy, apoplexy, or any of the forms of hysteria.

quite as much certainty, be affirmed that it is a state identical with that which strychnine is capable of producing. Now, this does not reveal to the most careful observation any appreciable departure from the normal state. I have repeatedly examined with the microscope the spinal cords of animals killed by strychnine, and have never been able to detect, even with the highest powers, the slightest change which I could regard as morbid.

The condition of the nervous centres in tetanus is, as it seems to me, best expressed by the term *exalted polarity*,—a condition which gives rise to the undue development of the nervous force. A peculiarity of this state is, that whilst the polar force of the nervous centre is at all times exalted, it is liable to frequent fits of augmentation or exacerbation, during which more extensive and powerful effects are produced. In this way we explain the apparent subsidence of the affection of the spinal cord for longer or shorter intervals, and the subsequent excitation of it in severe paroxysms.

If another similar case were to present itself, what treatment should we adopt? I do not know that I should pursue any different plan from that, which I have described to you in the case of our patient Franklin. I might, perhaps, give support earlier than I did in the present case. I should give quinine freely by the mouth or rectum: if

there were much difficulty of deglutition, I should throw up beef-tea enemata, and I should administer chloroform carefully. The administration of chloroform is objectionable on the ground that it is a depressing agent: now, in tetanus, you ought to husband and promote the strength of your patient as much as possible; the disease itself depresses more than enough; and an important aim in your treatment should be to enable your patient to sustain the reiterated shocks of the spasms: if, then, you give chloroform, take care at the same time to support him well, and be not afraid of giving stimulants—such as wine or brandy—if you think he can digest them, and especially if the difficulty of swallowing prevents the patient from taking more than a small quantity of nutriment at a time. The cases which I have known succeed the best, have all been treated with close attention to upholding the powers of the patient. If you bear in mind that the great danger is of death from exhaustion, and act accordingly, you will not go wrong in this particular. There can be no use in blood-letting in cases of this kind, as frequently practised formerly, nor in active purgation; the bowels, however, should be evacuated daily, or on alternate days, but more than this is likely to be prejudicial. Neither is there any good to be derived from giving those excessive doses of opium, that have been used in so many cases; for, besides the negative evidence of the usefulness of

this practice, there is this objection to it—that opium given in large doses tends to call forth that very exaltation of the polarity of the nervous system which we desire to combat. This is abundantly proved by experiments on cold-blooded animals. You may throw a frog into regular tetanus by opium; and, although actual tetanus cannot be produced by opium in warm-blooded animals, many circumstances tend to show that it may give rise to a state similar to, though much less in degree than, that which it can create in reptiles

Are we to expect any good from hydrocyanic acid? I think not: and my objections to it are much the same as those against opium,—viz., that it tends to exalt, and not to lower the polarity of the nervous centres. Those of you, who have seen the death of an animal from hydrocyanic acid, will at once appreciate this objection. You will remember the violent epilepsy—the tetanic epilepsy, if I may call it so,—the combination of tonic and clonic spasm, under which the animal suffers.

The use of galvanism has been proposed in cases of this description for the purpose of depolarizing the nervous centres, and has actually been tried by Matteucci. The long-continued passage of a current of galvanism tends to weaken and to paralyze a nerve or nervous centre, if the direction of the current be the same as that of the nervous force. In the case related by Matteucci, the spas-

modic condition of the muscles completely subsided during the passage of the electric current.

Our patient, Franklin, was treated in addition to the other means, by the application of cold over the region of the spine. Cold has a powerful effect in depolarizing the spinal cord. I have tried it with great advantage in this hospital in tetanus, in laryngismus, and in the convulsions of children. The best plan for its employment is to fill a bladder with some broken ice, and apply it directly to the spine: the ox gullets, from their length and cylindrical form, answer best; an intense degree of cold may be thus obtained in a very short time, and its effect on the circulation will soon be manifest, since cold has a very powerful influence in diminishing both the force and frequency of the heart's action: for this reason you must not apply it for too long a time, or over too great an extent of surface; you must watch your patient, and remove and re-apply it, as his condition shall indicate. In the cases, in which I have tried it, I have found it manifestly beneficial in diminishing the intensity of the spasms.

With regard to internal remedies, I know of nothing better than small doses of opium, belladonna, and conium. The two latter drugs certainly seem to me to have a depolarizing power, as is indicated by their influence, especially that of belladonna, in causing dilatation of the pupil. Still, a good depolarizing agent which does not

tend at the same time to weaken or depress the powers of the patient, is a great desideratum, and when discovered will no doubt prove an invaluable remedy in the treatment of tetanus. What, however, in the absence of such a remedy, the physician has principally to trust to, is support, in order that he may gain time, to enable his patient to weather the storm, to sustain the attacks of the disease, until the source of irritation shall have ceased to exist, and its consequences have abated.

I must not conclude without calling your attention to the great importance of isolating tetanic patients, and protecting them, not only from the excitement caused by many persons surrounding the bed, but also from the influence of draughts or currents of air, or external mechanical stimuli, which are peculiarly prone to provoke the tetanic spasm. These patients ought always, when possible, to be put into a separate ward, and visitors should be excluded. The temperature of the room should be kept at a moderate degree, and care should be taken to exclude sound, and also irregular currents of air and light, as far as it can be conveniently done.

LECTURE XIX.

On Cases of Chorea.

I MUST refer you to-day to several cases of chorea which have lately been in the hospital, to illustrate the remarks which I propose to make on the clinical history, pathology, and treatment of that disease.

CASE LXXVIII. The first case is that of a boy named May, in Rose ward (vol. xxxiii. p. 187), which affords an interesting example of the connexion of this malady with rheumatic fever. The history is as follows:—The patient is nine years of age; he is a thin, but otherwise healthy-looking boy; his friends are all quite healthy, and none of the family have ever been similarly affected.

We find that he had enjoyed very good health until ten weeks ago, when he was attacked with rheumatic fever; his ankles were first affected with the rheumatic swellings, and soon afterwards the knees, elbows, hands, and wrists. All these joints became very painful and much swollen; he also suffered from profuse sweats, and was affected

with a sense of weight and oppression about the chest. Under the treatment to which he was subjected (and this was by no means severe) he recovered; but for some time after the disappearance of the symptoms, the rheumatic state seemed to linger about him, until he began to exhibit signs of chorea, two weeks before his admission into the hospital.

The choreic symptoms seem to have come on in a rapid, almost sudden manner. The boy went to bed as well as usual, but in the morning, when his mother went to give him his breakfast, she was surprised to find that he could not hold his cup, and that he was quite helpless—indeed, much in the same state as he is at the present time, except that the symptoms were more limited to one side. He had lost the power of directing his movements properly; the motions of his limbs were exaggerated and ungovernable, and, if he attempted to take hold of anything, his arm appeared to be violently jerked, in the right direction perhaps, but usually beyond the object of his search, as if by some power over which he had no control.

Among the early symptoms which manifested themselves in this way was difficulty of deglutition, which came on and continued for some days prior to the more common and characteristic symptoms. The dysphagia was due partly to want of full controlling power over the tongue, and partly to a want of due harmony in the action of the pharyn-

geal muscles. This symptom is peculiarly interesting, from the marked connexion which subsists between this malady and emotional excitement; and it deserves your special attention in connexion with the pathology of this disease. Previous to his admission the boy could not walk or help himself in any way, and he was obliged to be washed and dressed by others.

On admission, he was unable to stand for any time without being supported; and if he attempted to walk or run, his limbs would be forcibly jerked about in various directions, and he would fall if not upheld. When in bed his arms and legs would be in constant restless motion, being thrust first to one side and then to the other, with an irregular jactitating movement.

His articulation was very imperfect, and if he attempted to speak he could only utter a few scarcely intelligible words in a whisper. The dysphagia, although much better, was still present.

When he was told to put out his tongue, it was protruded with the sudden thrust so characteristic of chorea, a feature of the disease, to which you will remember I have often called your attention, as one of its most constant and characteristic symptoms. The whole organ is forcibly and, generally, rapidly protruded from the mouth, appearing as if thrust between the teeth, and is retracted sometimes in an equally sudden manner, sometimes more slowly, and as if supported and guided by the teeth. There

is also an awkwardness and an exaggerated character in all the movements, which extend even to the muscles of the face. These phenomena are due to a want of power of duly co-ordinating the various voluntary movements of the body, and doubtless depend upon some affection of that portion of the nervous centres, in which the co-ordinating faculty is situated.

Another character of these irregular movements which, however, does not exist in this case, is, that they are more evident on one side of the body than on the other; you will find this feature evident in some of the other cases, which I shall presently relate to you. Chorea is very much a one-sided disease; that is, it affects one side more than the other; sometimes one side only is affected; hence it may be classed with cerebral affections, in which the morbid state of one side of the brain will extend to the opposite side of the body. It seldom happens, even although the irregular movements may exist on both sides, that we cannot detect more movement on one side than the other. The present case, however, seems to be an exception,—it is one of general chorea, and all the limbs seem equally disturbed in their movements.

What is especially interesting in the boy May, is the occurrence of the disease as a sequence to rheumatic fever: the case, too, affords a good example of the heart disease, which is so often associated

with chorea, and which, in this instance, is clearly dependent upon the rheumatic attack. Upon listening to the chest, a bellows-sound was readily detected, which was most distinct at the apex, and became less so as you receded towards the base of the heart; this bellows-sound being synchronous with the systole, and occupying the situation, which it did, must have depended upon such imperfection of the mitral valve, as allowed of regurgitation of the blood into the auricle at each contraction of the ventricle; it was also audible at the back, below the left scapula. The imperfection was probably the result of an endocarditis, which had formerly affected the mitral valve and the chordæ tendineæ, and created more or less of shrinking of the curtains of the valve and contraction of the cords, so as to prevent the perfect apposition of their margins, and the complete closure of the auriculo-ventricular opening.

This boy was taken ill with symptoms of chorea a fortnight before the date of his admission into the hospital, May the 8th. The treatment to which he has been subjected is sufficiently simple; it consisted merely of splashing with cold water every morning, and, at the same time, feeding him well. The splashing is effected by throwing one or more pailfuls of water over the patient, while he is standing in a large tub. This is a plan of treatment, which we have followed with benefit in several cases in the hospital within the

last few months; the effect of it in this case has been, that the boy, in about eighteen days, improved much in general nutrition, the irregular movements have diminished, and, at the present time, he is able to walk without assistance,—a result not more favourable, than that which attends its adoption in most cases. The bellows-sound remains, and probably will remain during the boy's life; for it depends upon organic imperfection of the valves, and must necessarily be present as long as that imperfection exists.

Another point in the history of this boy, to which I have not yet referred, is, the tendency in his family to rheumatic complaints; for we find that his elder brother, aged 14, also had an attack of rheumatic fever about a year ago.

A day or two before the symptoms appeared, this boy was much frightened by his sister, who had covered herself with a white sheet, and appeared before him unexpectedly, while he was in bed. There is here, then, that which we so frequently—indeed, I might say, so constantly,—observe, namely, the connexion of sudden fright with the origin of these cases. In this case the fright preceded the symptoms by a day or two; but, in some other of the cases to which I shall have to direct your attention, a much longer interval occurred between the fright and the manifestation of the symptoms. Although a certain

diathesis seems to be always present in cases of chorea, the disease seldom occurs without some sudden emotional excitement, such as fright.

Case LXXIX. The next case is that of Sophia Jackson, æt. 14, in Lonsdale ward (vol. xxxiii. p. 89); she is one of two sisters, both of whom have suffered from chorea. She had always enjoyed good health, with the exception of the occurrence of an attack of rheumatic fever, when she was two and a half years old. About a week before her admission, it was noticed that her hands became fidgety, and this was especially the case with the right hand. In this case, the one-sided character of the disease is well illustrated, for the irregular movements are almost confined to the right half of the body.

Here, also, we have fright in the history; we find that, about three weeks before the appearance of the symptoms, she was met and accosted by a drunken man, and was very much alarmed at the time; but you will observe that, in this case, so long an interval as three weeks elapsed between the occurrence of the fright and the manifestation of the characteristic symptoms. Although this interval seems very long, I think we may fairly refer the excitement of the disease to this cause; in many instances, indeed, even longer periods have elapsed between the fright and the accession of the malady; and it is very rare

indeed to meet with a case, in which you may not find, on investigating the history, that there has been a fright from some cause or another. I have known it occur six weeks before the chorea manifested itself.

The mobility of the right upper extremity continued to increase in our patient, and soon the power of walking became obviously impaired. The peculiar thrust of the tongue was also developed; and, upon examining the heart soon after her admission, we detected a bellows-sound over the base of the organ, and in the course of the large arteries. This was most probably due to the anæmic state of the patient, and, therefore, in this point differed materially from the bellows-sound present in the first case; this anæmic murmur disappeared as soon as she recovered. In this case we noticed a feature of not unfrequent occurrence, namely, that the urine was of high specific gravity, and generally contained a deposit of pale lithates, and an excess of urea was found in it several times. The high density of the urine was most marked when the choreic movements were most active, and it might, in part at least, be attributed to the increased waste of tissue consequent on the disturbed state of the muscles and nerves.

This patient was ordered an affusion of cold water every morning, and was kept upon a good, nutritious diet. The urine was carefully examined

from day to day by Mr. Liddon; its specific gravity never fell below 1019, and frequently reached 1030, and once was found to be as high as 1035. As the patient improved in health, the urine fell in specific gravity, but was never found below 1019. Lithate of ammonia was nearly always present, and oxalate of lime was frequently found mixed with it.

This patient was admitted on the 6th of March, and discharged quite well on the 12th of April.

Case LXXX. The next case is of a much more serious character than that which I have just related. Mary Jackson (sister to the last patient), aged 12 (vol. xxxi. p. 160), was admitted on the 29th of November, 1850, and was a long time in the hospital before she recovered. Her parents and the other members of her family are healthy, with the exception of the sister, to whose case I have just now referred.

The history of this case, as taken by Mr. W. Brown, is as follows:—Nine weeks before her admission she was frightened, as her sister had been, by a drunken man; a few days after this she felt pain in the right arm and leg, and experienced a tingling in the fingers, which became restless and fidgety. Soon after, the left extremities became similarly affected; then came twitchings in the face; and, in the course of a fortnight from the time of the fright, the irregular

movements became general, she lost the power of standing or walking, and her articulation was almost completely destroyed. She was in this state, when admitted into the hospital. The jactitating movements were extremely well marked; and you will remember that I often called your attention at the bedside to the striking contrast which the irregular jerking clonic convulsions of the muscles exhibited, when compared with the tonic spasms of tetanus. As is always the case in the more aggravated instances of this disease, any attempt to restrain the movements, as by forcibly holding down the legs or arms, invariably tended to their aggravation, and excited them very much in some other part of the body which had not been subjected to restraint. From the jerking movement of the muscles, the pulse could not be distinctly felt. The heart's action was extremely rapid—136, but varied as to rate of movement; and there was a decided mitral bellows-sound.

It is of some interest to notice, that this girl was the second in the same family who was affected with chorea. The two children are of very similar make and habit; their nervous systems are alike; and, probably, their power of generating the nervous force is similar. It is thus that chorea often attacks members of the same family who are of like build and constitution, and have been equally exposed to emotional excitement. But it cannot be said

that chorea is hereditary, *i. e.*, handed down from parent to child.

The treatment pursued in this case was just that, which had been adopted in the others, namely, at first the splashing with cold water night and morning, and the subsequent administration of quinine and iron, with occasional aperients. The effects of this treatment were soon manifest. It was commenced on the 30th of November; on the 3rd of December the movements had become much less, and the heart's action was greatly reduced in frequency; on the 6th she was so much quieter, that the pulse could be counted at the wrist, and it numbered 74; on the eighth, she was ordered a grain of quinine thrice a day; and, on the 14th, citrate of iron was substituted for this in the dose of three grains thrice a day; on the 17th, she began to walk with the assistance of the nurse; and, on the 20th, she was able to walk without assistance. She now began to use her hands, and could grasp feebly, but not so well with the right as with the left. She gradually but slowly improved under the use of steel, with occasional purgatives, good diet, and cold splashing; but it was not until after the expiration of the long period, of upwards of three months from her admission, and of five months from the invasion of the disease, that she was discharged quite cured.

During the first three weeks of the treatment of

this case, the specific gravity of the urine was very high; 1040, 1037, 1030; it afterwards fell to 1020. The mitral bellows-sound had not disappeared when she left the hospital.

Case LXXXI.—The next case is that of Emma Skinner, aged 10, who was admitted April 12th (vol. xxxii. p. 122); she is about ten years of age, the period of life at which chorea is most apt to occur. Chorea is really a disease of childhood; and although we sometimes see symptoms somewhat resembling those of chorea at the adult period, and at more advanced ages, such cases are clearly exceptional; and it may be questioned, whether they are due to exactly the same morbid condition as that which gives rise to the ordinary choreic convulsions of early life. Chorea generally occurs between the ages of 9 and 15; but, as I have just stated, we occasionally meet with it in patients a few years younger or older, and sometimes symptoms of a similar kind occur in persons of advanced age.

This case illustrates a point which you should bear in mind in reference to chorea, and many other diseases of the same class,—I mean, the great disposition of the malady to recur. A child, who has had one attack of chorea, is always liable to the occurrence of a second at some future period. The same tendency is met with in all paroxysmal diseases. It appears that the patient in this case

has been on two former occasions in this hospital for chorea. The present attack came on about three weeks ago, when she was recovering from scarlet fever, at which time she was no doubt in a state of very enfeebled nutrition, a condition peculiarly favourable to the manifestation of chorea. There is no evidence that any distinct fright occurred in this particular instance; in the every-day life of most children, and especially of those of the lower classes, there are numerous causes of mental emotion, which, although trifling and transient, are amply sufficient to excite nervous disturbance in a weak child. Much the same symptoms were present in this as in the other cases; the choreic movements were strictly limited to the left side, and the case afforded a striking illustration of the one-sided character, which the disease is so apt to assume, the right side being wholly free.

The urine also was of very high specific gravity, and frequently deposited pale lithates in considerable quantity. This deposit of lithates is, as I have mentioned, very common, and likewise of grains of lithic acid, which are often as numerous and as large as we see them in adults of gouty or rheumatic habit. Crystals of lithic acid also frequently present themselves on a microscopical examination of the urine, among which are often found crystals of oxalate of lime in great numbers. Generally speaking, the density of the

urine is highest in those cases in which the movements are most general and most active; and it falls steadily with their diminution and with the restoration of a greater controlling power on the part of the patient.

You cannot fail to notice, that the general character of the urine in chorea bears a marked resemblance to that in rheumatism. This and other circumstances have led me to associate the state of constitution, in which chorea is apt to occur, with the rheumatic diathesis. The proneness of patients of rheumatic constitution to attacks of chorea, the tendency to a repetition of the attacks in both diseases, the frequency in chorea of endocardial affections, so common in rheumatic states, all point to this affinity between the two disorders.

The four cases to which I have now directed your attention give you a good view of the clinical history of chorea. What is the nature of this malady?

It is easier to say what chorea is not, than to describe what its essential nature is. We may regard it as a disease dependent on a debilitated state of the system, which does not in any way arise from an inflammatory or hyperæmic state of any part of the great nervous centres or of other organs. Indeed, it is impossible to fix upon any particular organ of the body, in which anything like structural lesion exists as a constant feature, in cases of chorea. The disease is one of functional dis-

turbance, rather than of organic change; and this is borne out by the results of *post-mortem* examinations; for almost without exception we fail to detect, in those cases of chorea which terminate fatally, any morbid alteration which, physiologically, could give rise to the phenomena; and in the vast majority of cases, we find all the viscera in a perfectly healthy condition, at least so far as we are enabled to make out with the means at present at our command.

The structures, which are obviously affected in chorea, are the nerves and muscles. Doubtless a morbid state of both exists; but it seems most probable that the disturbed state of the muscles is excited and maintained by a deranged state of the nerves and nervous centres. If I were to refer to any particular part of the brain, as more particularly the seat of that disturbance, which gives rise to the development of the peculiar phenomena of chorea, it would be to that, which may be regarded as the centre of emotion. The remarkable frequency with which the attack of chorea is traceable to fright as its cause, points clearly to this part of the brain, (which has the most extensive connexions with, and influence over, other parts of the nervous system,) as the *primum movens* in the production of choreic convulsions. The chain of phenomena would then be as follows: first, a peculiar diathesis, then a more or less enfeebled nutrition; thirdly, a strong mental

impression, which disturbs the centre of emotion, and, through it, deranges the action of more or less of the nervous system, and of a corresponding portion of the muscular system.

And now as to the treatment. You will readily admit that it is not a little important to have it impressed on the mind of the practitioner, *in limine*, that he has not to combat any inflammatory, or hypersthenic, or disorganising process. He has simply to improve the condition of the blood, to amend general nutrition, to calm the nerves, and to infuse tone into the muscles. Of course, if any source of irritation exist it must be removed, such as a deranged state of bowels or worms in the intestinal canal. These causes of irritation, however, occur much less frequently than is generally supposed. Nevertheless, they deserve early and close attention; but a system of purgation carried on too long may prove highly pernicious. If worms be present, let them be expelled if possible, whether they be regarded as cause, or simply as concomitant. The most common of these are the ascarides, which have their habitat in the rectum, and which may be expelled by enemata of a strong solution of salt in water or in an infusion of quassia.

Attention to the fulfilment of these indications is all that is essential to the cure of the vast majority of the cases of chorea, which come before us in practice. But you will find great advantage,

in most instances, from the free use of cold water by affusion to the surface of the body. I have treated many cases most successfully, simply by attention to diet, and by cold affusion, practised once, twice, or thrice a day, and without the use of drugs of any kind. You will remember, that this practice was followed very speedily by marked benefit, in the third of the cases which I have detailed to you to-day. The girl had been under tonic treatment, for two months prior to her admission, with but little benefit; but on the fourth day of the use of the cold affusion there was a very marked improvement in both the frequency and the violence of the movements; and it was evident that the affusion exercised a decidedly calmative influence upon the nervous system.

Sometimes, however, you will find yourselves balked in the use of this valuable curative agent, in the case of an irritable, or excitable, or ill-tempered child, or of a nervous fidgety mother, or a prejudiced nurse. When you have to contend with these difficulties, you must be guided by circumstances, either in giving up the resource, or in gradually leading the patient to the full use of it.

I wish to impress upon you that it is to such hygienic treatment as I have described, that we must look *mainly* for the cure of chorea. But there can be no doubt that much good may be obtained from the simultaneous and careful employment of drugs of the tonic class. Of these

the metallic tonics rank first, especially iron and zinc, or quinine, or some form of bark, of which you will find the liquor cinchonæ of Mr. Battley one of the most useful preparations, or mineral acids; sometimes also cod's liver oil, and sarsaparilla.

There are certain after-effects of chorea sometimes met with, which you should remember, and be prepared to treat. A child may be brought to you with one side completely paralysed, or with palsy of a limb, and, upon questioning the friends, you will find that the child has been left in this condition after an attack of chorea.* Occasionally this paralytic state occurs as a precursor to the choreic attack, but this is rare. What are you to do in such a state? Does this condition indicate the occurrence of any important lesion? In my experience certainly not; most of the cases get well in a short time. You should persist in the tonic plan of treatment, and endeavour to improve the health as much as possible. If, however, the paralytic state should last for any time, it is important to use means to exercise the paralysed muscles, otherwise they would suffer in nutrition, and a permanently weakened state of the muscles would result. For this purpose, nothing is better than the careful use of galvanism. A gentle and

* Vid. ante, Lect. XV.

slowly-interrupted current from the battery may be daily transmitted through the paralysed limb or limbs, for a quarter of an hour or twenty minutes at a time; and the limbs may be frequently rubbed with a coarse towel or a flesh-brush.

The prognosis may almost always be favourable in chorea, save in those terribly acute and general cases which, by the violence of their movements, rapidly exhaust the powers of life. But, even in such cases as these, we shall be more successful, when we can succeed in divesting ourselves of the notion, that such symptoms can only arise from a hypersthenic or inflammatory state. I may here mention, by the way, that the cold affusion, well managed, and repeatedly applied at short intervals, has great influence in controlling the convulsions in these cases of general chorea.

It is not easy to answer a question which is often asked, especially in private practice,—namely, as to the probable duration of a choreic attack. Generally speaking, the restoration to health, in such cases, is a slow and tedious process; and, I think, you will find it more so in private than in hospital practice, because we cannot subject our patients to the same discipline, when they are exposed to the excitements and indulgences of home life, as when under the surveillance and direction of strangers, in a well-regulated public establishment. It is a very mild case of chorea

which will recover in three or four weeks; more frequently they require double that time,—often two or three months, or even five or six months, as in the case of Mary Jackson.

LECTURE XX.

On local Hysteria, and on Catalepsy.

Let me call your attention to-day to two interesting cases of nervous affection. The subject of the first is still in the house, but that of the other case left it a few days ago. I regret that, in consequence of the transitory nature of the symptoms from which this second patient suffered, and from the attack having occurred at midnight, few of you had an opportunity of witnessing it. Her symptoms were of a very interesting nature, and not often met with. For these reasons, and notwithstanding that so many of you missed seeing the case, I think it may, in some degree, supply the failure of this opportunity, if I detail the case to you, and make some remarks on its pathology and treatment.

Case LXXXII. First then, we take the case of Harriet B——, aged 30, in Lonsdale ward, for the notes of which we are indebted to my clinical clerk, Mr. (now Dr.) T. Bridgwater (vol. xxxiii. p. 177). This patient was admitted on the 7th of May, 1851. The main point of interest about the case is, that she suffered acute pain in the

right hypogastric region of the abdomen; the pain was so acute that she could not bear the slightest pressure on the affected part; even the weight of the bed-clothes was painful to her. This pain was her chief complaint, and for the relief of this she was admitted to the hospital. It would be impossible to find an instance of a more strictly localised pain than that afforded by this case, nor could I bring before you a more striking example, in which relief from pain was peremptorily demanded from the physician, as the one thing complained of by the patient. Cases of this kind you will often meet with hereafter in your practice, and you will do well to note carefully such as may come before you now, and the treatment which may appear to be serviceable; for nothing contributes to professional success more than the power of readily appreciating the nature and causes of pain, and tact in applying suitable and efficacious remedies.

Severe abdominal pain, whether general or limited to a spot, is at once suggestive, both to practitioner and patient, of inflammation within the abdomen; it may be, of peritonitis. This latter malady is, as you know, a severe one, and, in the majority of cases, fatal. How important, then, is it to be able to determine whether such pain is due to a cause of this kind or to some other!

The pain under which this woman suffers is

referred to the right hypogastric region, over the position of the right ovary, and the painful part occupies a circular space of about two and a half or three inches in diameter. There is extreme tenderness to the touch; even when the integuments are pinched up with the utmost gentleness, and with every care to avoid compression of the subjacent parts, she shrinks and appears to suffer great pain. *Deep* pressure on the corresponding region of the left side causes pain likewise, as if by implicating the right side.

There is another very important feature in the case which deserves especial notice; it is this, that when her attention is much engaged, she certainly suffers less, although she cannot be said to be free from pain. Upon making a careful examination of the part, we could detect no tumour; and, upon pressing deeply, no abnormal condition of the ovary could be discovered; and, although there was pain, there was no increase of it to a proportionate extent.

Her history we found to be as follows:—She is unmarried; but, many years ago, she fell a victim to the seduction of some improper person, and, having become pregnant, she suffered a very severe labour, in which the perinæum was lacerated; in consequence of this injury, she was under surgical treatment in the hospital for some time.

Three months before her admission into the hospital, she had a severe attack of diarrhœa, ac-

companied by vomiting. Her bowels have since continued in an irritable state. The catamenia have always been irregular, and there has been a constant drain, more or less, from leucorrhœa, which increased in quantity as each menstrual period recurred.

On her admission into the hospital, she was still suffering from diarrhœa, but there was nothing very unhealthy in her expression of countenance, nor was her abdomen unduly swelled. The abdominal walls were lax, and the bowels were but slightly tympanitic. It appeared that the pain came on at the same time as the diarrhœa, and at first was aggravated by taking food.

This curious and not easily explicable connexion between the hypogastric pain and the state of the alimentary canal, led me to direct the early treatment to allay irritation by checking diarrhœa, which, if allowed to continue, would have impaired her general health, and aggravated the other symptoms under which she laboured. With this view, then, she was ordered to take astringents with opium, and under the influence of these remedies the diarrhœa was checked, but the abdominal pain and tenderness remained as before; hence it became the more important to determine the precise nature of this pain; and, with this view, let me conduct you through the various steps of the diagnosis.

We will suppose ourselves at the patient's bed-

side; and, having attentively listened to her history, and heard her complaints, we find that she suffers great pain upon the slightest pressure in the right hypogastric region, in the position and over a space such as I have already described. Upon further examination, we find the pain nearly as great under slight, as under deep and heavy pressure.

What, then, are the conditions that might give rise to such a pain as this?

The first and most obvious cause, which the history at once suggested, was that the pain had its seat in the irritated bowels. It was in the region of the termination of the ileum and commencement of the colon; and there is no cause so fruitful of diarrhœa as irritation of those parts of the intestinal canal. But the objections to this view of the case were as follow. There was no tympanitic state, such as bowel irritation sufficient to create so much pain would infallibly produce. Again, the bowel irritation was easily controlled very soon after her admission into the hospital, yet the pain remained in all its intensity.

Secondly, it might have been due to a local peritonitis; but the objections which I have just urged to the pain having its seat in an irritated bowel apply with equal force to peritonitis. And, as an additional objection, also applicable to both views, I may here state the fact that the pain was as great when the integuments were gently

pinched up, as when deep pressure was made; whereas, in peritonitis as well as in inflamed bowel, firm and deep pressure would cause by far the greatest amount of pain.

A third cause might have been inflammation of the ovary. To this view, however, was opposed the absence of any swelling or tumour in the hypogastric region, and of increased pain on deep pressure. And, on examination *per vaginam*, Dr. A. Farre could not detect anything wrong with either ovaries or uterus.

Any inflammatory affection of the abdominal muscles, or of the iliacus internus or psoas muscles, was insufficient to explain the pain, because the movements of the trunk or of the limbs could be effected without pain; and, indeed, with all three of these conditions it may be regarded as quite incompatible, that our patient had never had, since she came into the hospital, that amount of fever, which would accompany a severe internal inflammation.

Lastly, the symptoms might be referable to the peculiar state of the nerves, superficial as well as deep, which gives rise to that kind of pain which conventionally (and, in the present state of our knowledge, conveniently enough) we call "hysterical pain." This pain, also called a nervous pain, is no doubt just as acute and severe to the patient's feelings, as any pain from injury or organic lesion, although, in the parts in which it is

seated, we can discover no alteration of structure whatever.

You see that we have excluded from the diagnosis peritonitis, enteritis, and inflammation of the ovary. Let us now inquire how far the hypothesis of the pain being hysterical is borne out by the accompanying phenomena.

The great tenderness of the skin to the slightest touch is very favourable to this view, which is likewise supported by the fact of the little difference in the intensity of the pain, under deep and under superficial pressure.

The slightest touch to any other part of the body makes the patient suddenly shrink back with a greater or less expression of pain,—a character which belongs to all these hysterical affections. Often when you bring the finger close to the skin, there is an expression of pain nearly as great, as if you had actually touched it.

Certain features in the general constitution of the patient confirm this view; she is evidently of an hysterical constitution, and exhibits that peculiar appearance of countenance, which I have often pointed out to you by the name of "facies hysterica," characterized by a remarkable depth and prominent fulness, with more or less thickness, of the upper lip. There is also a fulness and obviously drooping condition of the upper eyelids. This drooping conformation of eyelids is at once a mark of beauty, and of that from which many

beautiful women suffer very much, namely, the hysterical state of constitution.

She also exhibits that irritable state of spine, under which hysterical patients are apt to suffer. The least pressure on a spinous process causes her to shrink, and to complain of a pain shooting forwards from the point pressed on; and, as is so often the case with patients of this temperament, she complains of pain from pressure, in whatever part of the body it may be applied; wherever she is touched, whether the finger comes in contact with her arms, or back, or any other part of the body, she exhibits an undue degree of sensibility, and shrinks.

Then there is yet another point about the case, which I have not mentioned. We find, on referring to the history, that the patient never menstruated regularly. In the vast majority of cases of hysterical affections you will find something wrong about the uterine function; and, although I am not prepared to lay it down positively, that the disease (as its name implies) depends upon this disturbance, you will find in the many forms of hysterical affections that may be brought under your notice, that it will rarely happen that there is not some deviation from the healthy action of the uterus exhibited in the disturbance of the menstrual function. A large proportion of hysterical patients suffer from leucorrhœa; in others the catamenia are insufficient in quantity, or

occur at irregular intervals. Sometimes the menstrual secretion recurs at short intervals, and is very profuse, and we have that condition to which the term "menorrhagia" has been applied. Perhaps there is an irritable condition of the uterus, causing almost constant pain and tenderness in the uterine region. No one of these conditions is constantly observed, but in almost every case of hysteria there is some form or forms of uterine disturbance.

Thus, then, we arrive at the conclusion, that the symptoms in Harriet B——'s case are to be referred to an hysterical state of constitution. The case is a good example of one form of local hysteria, to some of the other forms of which I shall briefly call your attention.

Of all these conditions, that which has been called "irritable uterus," is by far the most formidable. It is characterized by exquisite pain and tenderness in the region of the uterus,—even the slightest pressure over the organ, or the least touch applied to it in the examination *per vaginam*, causes great suffering, and the patient shrinks in consequence. At the same time there is no enlargement or other organic change in the organ.

Another form of local hysteria is a pain of very frequent occurrence under either breast, and, what is rather curious, more commonly found under the left than under the right breast. Leucorrhœa

is so often concomitant with this pain, that whenever a patient complains of it, I invariably ask if she suffers from leucorrhœa; or conversely, if leucorrhœa be present in any quantity and for any time, I am led to inquire about the local pain. That there is a connexion between the leucorrhœa and the pain in the side scarcely admits of a doubt; for the pain is more severe if the leucorrhœa be profuse, and it very commonly happens, that as soon as the healthy condition of the uterine functions has been restored, the pain will disappear. My theory of the production of this pain is this: I believe that in cases of this kind there is not only an irritated state of uterus, but also of one or both ovaries, and the pain is more immediately associated with the irritable ovary, the nerves of which, implanted, as many of them are, in the spinal cord, reflect the irritated state on to the nerves of the submammary region. It is an interesting example of a reflected sensation.

A third form of local hysteria is one with which you should be familiar, and able readily to diagnose. It manifests itself in that pain in the stomach, which is commonly called "gastrodynia." We must be very careful to distinguish this from the pain resulting from ulcer of the stomach, as an essentially different treatment would be applicable to each. One form of ulcer of the stomach very often occurs in young chlorotic women,

amongst whom also we most frequently meet with the gastrodynia; hence the importance of paying close attention to the symptoms, which will enable us to distinguish with certainty the one from the other. An important difference is, that in the hysterical affection, the pain may occur at any or at all times, whether the stomach be empty or full; nor is there any constant relation between its development and the ingestion of any particular kind of food. On the other hand, the pain from ulcer of the stomach is distinctly influenced by the taking of food, but especially of that kind which it is the province of the stomach to digest. Patients with ulcer will tell you, that as long as they abstain from food, they have little or no pain; but as soon as they eat anything they begin to suffer, and they are not easy until digestion has been completed. Hysterical patients, on the other hand, will tell you, that the pain comes on as soon as they rise in the morning, and continues perhaps all day. You will also find that these patients exhibit more or less of the hysterical constitution. With reference to the diagnosis between these two kinds of stomach pain, you should always inquire if there has been vomiting of blood, as this forms a most important symptom in the history of ulcer of the stomach. Or, if there have been no vomiting of blood, you should inquire whether it may not have passed through the pylorus into the intestinal canal, in which case

you will find upon inquiry, that the patient has passed very dark matter from the bowels; the stools, in fact, exhibit a pitchy character in those cases in which blood from the stomach passes by the bowel. The effects of the treatment to which the patient is subjected will also often enable you to distinguish the true ulcer of the stomach. Beer and stimulants, and all acid drinks, greatly aggravate the pain from ulcer, while they often tend to relieve the hysterical affection. Bismuth acts very favourably in the latter, but not so well in the former.

Another form in which we find local hysteria manifesting itself is exhibited in that condition called "hysterical spine." You have a good example of it in the patient whose case has led to these remarks. If you pass your finger down the spine, you will find some places very irritable; perhaps the painful situation may be confined to one particular spot, or it may extend over three or four spinous processes, or the whole spine may be affected. Wherever it be, you will find that the patient suddenly shrinks as soon as the irritable part is touched, and appears to suffer, and no doubt does suffer, exquisite pain. Now, this condition has been over and over again mistaken by careless practitioners for disease of one or more of the vertebræ; and in consequence the unhappy patients have suffered from all the artillery of physic; leeches, blisters, and setons have been

applied to the spine, and other antiphlogistic measures have been resorted to, but without any effect, beyond, perhaps, aggravation of the pain. The most important point, by means of which you may distinguish vertebral caries from the hysterical affection, is this: in the vertebral disease the pain is not so excessive, and is always fixed in one part, and it will be found to increase gradually as the disease advances.

Hysteria likewise affects joints. A patient has a pain in her knee or her hip, or some other joint. I have no doubt that in these cases there is not only an affection of the sentient but of the muscular nerves likewise; for the muscles are either spasmodically affected, or relaxed, or paralysed, occasioning difficulty and awkwardness in the movements of the joint.

Many instances of what are called neuralgic pains are referable to the hysterical state, and the case of Harriet B—— is one of them. But you must take care to distinguish the true neuralgic from the hysterical affection. They are different diseases, although probably nearly allied. In the true neuralgia there is an altered nutrition of the affected nerve as decided as that of the tissues of a joint affected with a transient attack of gout. Such, however, is probably not the case in hysteria.

Our patient, Harriet B——, has continued in the hospital for a considerable time. Repeated

examinations tended only to confirm the diagnosis I have given, and she has had two or three hysterical fits. On her admission she was treated for the diarrhœa under which she was then suffering. The diarrhœa ceased, but the pain remained unabated.

Since that time the treatment has been chiefly such as would check leucorrhœa, improve tone, and promote general health. The tepid shower-bath, the cold hip-bath, mild aperients with galbanum, and the citrate of iron, were administered for some time. The result was a very marked improvement of the general health.

Notwithstanding this improvement the pain in the hypogastrium continued troublesome, although diminished in severity. Local treatment was employed; leeches, blisters, and opiate applications were applied, without good effect. At length, fearing that these applications tended rather to aggravate the pain, by fixing her attention upon the affected part, as undoubtedly they are apt to do, I applied a blister to the corresponding spot on the left side, and kept it discharging for some time. This treatment was attended with the happiest result; the pain left the right side, but, as if in illustration of its true nature, she now complains of a pain in the left or blistered side, less severe, however, than the original pain. But from the great improvement in her general health, and the abatement which has already taken place

in her symptoms, there can be no doubt that time is now the most important element in her cure.*

Case LXXXIII. I shall now proceed to the second case; it is, I think, well worthy of your attentive consideration. The patient's name is Amelia D——, and she is 38 years of age. (Vol. xxxiv., p. 64.) I am sorry that she is not still in the hospital. The fact is, that the urgent symptoms for which she was admitted yielded so quickly, that she remained in the house only a very short time. Still, the symptoms were so remarkable, and the case so rare, that I feel I should not be doing right were I not to bring it specially before you.

In giving you a history of this case, I cannot do better than quote from the graphic description in the notes made by Dr. Hyde Salter, who, as house-physician, saw her from the moment of her admission.

"On June 5th, about ten o'clock in the evening, (Dr. Salter states,) I was called to a patient just brought into the hospital by a policeman, who had found her speechless in the streets. I saw a woman sitting in a chair, looking

* This patient remained in the hospital till August 31, and was then discharged very much improved in every respect.

quite intelligent and coherent, although evidently in great excitement and distress; her expression was anxious, and she looked from one to the other in an inquiring and imploring manner; her teeth were fast clenched, and her lips parted. On being asked who she was, she shook her head; when asked what was the matter with her, she pointed to her mouth and masseter muscles; when asked if her jaws were locked, she nodded her head. I then tried with all my force to separate them, but could not; then I felt her cheeks over the masseters, and found these muscles contracted into hard knots, which sufficiently explained the closure of the jaws. When questioned as to the cause of the tetanus, she clenched her fist and struck her left cheek, implying that she had had a blow there. I said, 'Have you had a blow there?' She nodded her head. We asked if she could write. She shook her head, and clasped her hands in a despairing way. We then asked her if her husband had given her the blow. She nodded assent eagerly.

"By degrees, putting all sorts of questions, and getting nods or shakes of the head, as the case might be, I learned that she lived in the neighbourhood of London, that she had been struck by her husband that day at two o'clock, and that the blow was the cause of her illness; also, that she was thirty-eight years of age. All these things afterwards proved to be true. While

answering our questions as well as she could, she suddenly, and without any warning, slid from her chair in a state of apparently complete insensibility, and would have fallen on the floor had she not been supported. Then commenced a series of spasms more strange and horrible than can be conceived or described; tetanic and clonic, partial and complete, symmetrical and irregular, varied in every conceivable and inconceivable way. First she had opisthotonos, then she was thrown forwards, then she twisted round and writhed like an eel, then she would throw herself forward and raise herself into a sitting posture, then she would roll over and over, then a slow undulation or wave of spasm would pass over her from head to foot, producing different movements of her limbs as it passed down, like a dog dying of hydrocyanic acid poisoning."

You might almost be led to think that this description is overdrawn, but I can myself bear testimony to the accurate statement of the facts, as I was present part of the time. Then, continues Dr. Salter, "the tonic spasm would suddenly become clonic, and she would throw out her arms and draw them back with great force. The legs would be affected in a similar way, or drawn up to the body, so that the heels were close to the buttocks, or thrust stiffly out. When holding her hand, she would suddenly clutch mine with such force that I could not disengage it. Meanwhile, her face was undergoing all sorts of con-

tortions, at one time expressing rage, at another intense fear, then a sneer, then a fixed and rigid stare; the eyes might be rolled upwards or downwards; the pupils dilated, and insensible to light; then the elevators of the upper lip and ala of the nose would jerk, perhaps on one side or both, exposing the teeth. The depressor anguli oris would draw one corner of the mouth down on the chin, or the platysma throw the skin into a state of rigidity;" but, as Dr. Salter remarks, it is quite impossible by description to convey any accurate idea of the extraordinary contortions of the patient.

One of the most remarkable features in the case was the rigid tonic condition of the whole muscular system. Every muscle employed in any movement became at the same time as hard as a board, and the movement was not so much executed, with rapidity, as with great force and even slowness. But in watching her attentively, it was plain that the movements, varied and irregular as they were, did not partake strictly of the character of involuntary movements. They were evidently influenced by a will, but by a diseased and an ill-directed will. While these spastic movements of the muscles continued, there appeared to be complete insensibility to surrounding objects; but when they ceased, her intellect seemed quickly to recover itself, and she could talk freely and collectedly. At this time she gave us her history, which we afterwards

ascertained to be correct. In the midst of her narrative, her eyes suddenly became fixed, and she stared at us for a short time and fell off into another paroxysm, and went through the same series of varied movements and contortions as before. She continued in this state, alternately in fits and quiet, for nearly two hours, and then slept.

Now, on being called to a case of this kind, you would have, on the spot, to decide upon its real nature, without the help of any history on which full reliance could be placed. Such was our case, when we first saw this patient; and, therefore, before I give you any further details, I shall state to you the conclusion arrived at by Dr. Salter and myself, and the grounds of our decision. We had to distinguish these extraordinary muscular movements from those of trismus, of tetanus, and of epilepsy.

The distinction from trismus was sufficiently easy. The fact of her coming to herself so soon, and the subsidence of the spasmodic action of the muscles, were quite sufficient to enable us to determine, that the condition was not trismus, and the absence, in the history, of all those causes which ordinarily give rise to this affection, also tended to the same conclusion. In the same way that we were enabled to say, that we were not dealing with a case of trismus, we also came to the conclusion that it was not one of tetanus.

Trismus and tetanus are but degrees of the same affection. In both diseases the muscular movements are distinctly involuntary, and often excited by the application of some stimulus to the surface. In this case, however, the movements bore a decided resemblance to the voluntary class, and could not be excited at the will of the observer.

At first sight, the affection bore a greater resemblance to epilepsy than to any of the other forms of convulsive disease; but there were certain points which enabled us very positively to decide that it was not epilepsy. These were the nature of the movements, and the affection of the consciousness. The movements partook more of the tonic than of the clonic character; and although at times very much varied, they were combined and regular, and directed to an end and by a purpose, rather than irregular, rapid, and, as it were, explosive, as is the case with the convulsions of epilepsy.

The most important feature was the state of the consciousness, and by this chiefly we were enabled to decide against the epileptic nature of the case.

In epilepsy, complete coma, with total loss of consciousness, forms an essential and specific character. Convulsions form no necessary part of the epileptic paroxysm. There may be in epilepsy a complete absence of convulsions, but the insensibility is invariably present. Thus we often meet with instances in which there is sudden and even

momentary loss of consciousness, without any convulsion whatever. Such cases are not less truly examples of the epileptic paroxysm, than if the most violent convulsions were also present. Attacks of this non-convulsive kind constitute what the French call *petit mal;* but, in many instances, it is rather the *grand mal,* for the brain and the intellectual powers suffer much more after repeated attacks of this nature than after fits, in which the convulsive movements form the prominent feature of the disease.

In the case before us, however, we had no complete loss of consciousness; there was, undoubtedly, an affection of consciousness, that in which a person may be insensible to all ordinary external stimuli, and quite without the perception of what was going on around her, but at the same time aware of the altered state of the mental powers. In epilepsy the patient knows nothing of his mental state during the paroxysm—he is only conscious of his state before and after it—and the period of the fit is to him as if it had never existed.

The nature of the movements showed that consciousness was not wholly in abeyance; they exhibited a co-ordination and a regularity, which we never observe where there is complete loss of consciousness. She bent her fingers in a steady regular manner, as if the muscles were obeying the mandates of her will, and her body was contorted by a combined action, forming a striking contrast

with the jerking movements, which we see in a state of insensibility. I say, then, that in this and in other cases of a similar nature, we have no complete loss of consciousness; the apparent insensibility depends upon an intense concentration of the attention on one particular object. Let me illustrate what I mean in this way. We all know that when the mind is very much interested and occupied with any particular subject or train of thought, one is very apt to take no notice of occurrences which are taking place close to, or in immediate connexion with one's person; and to be unconscious even of noises; nay, one may suffer a moderately severe pinch, without being sufficiently roused from the state of reverie to direct attention to any but the one object; in fact, the mind is fixed on that one object to the complete exclusion of everything else for the time.

The state of consciousness into which such patients as Amelia D—— fall is of this nature, although very different in degree. What is commonly called "absence of mind" is an analogous affection of consciousness. The attacks are generally brought on by some powerful mental emotion—grief, anger, jealousy—which overwhelms and suspends all other intellectual operations, while its influence lasts.

Then, if the disease were not trismus, nor tetanus, nor epilepsy, what was it? All the symptoms and the history of the case combine to

show that it was an example of a highly-developed hysterical paroxysm or fit, or more exactly, of that peculiar and aggravated form of hysteria, which is called *catalepsy*.

The history, which has been carefully collected and recorded by my clinical clerk, Mr. Maurice Davis, shows that our patient was a highly-excitable, hysterical person, who has been subjected to moral, and perhaps physical influences also, well calculated to keep up that state. She tells us that she enjoyed good health, until she reached the age of ten, when she experienced a sudden fright, and fell, in a fit, two storeys down a well-staircase; from that time until her marriage (at the age of 15), she had the fits repeatedly, generally twice a week, and sometimes oftener. Since her marriage, she has had the fits less frequently, but has never been entirely free from them.

It is difficult to determine what was the precise nature of these fits, whether they were epileptic or hysterical. Her age was more favourable to the former; but the subsequent history, and the fact elicited from her, that she was advised to marry in order to get rid of the fits, rather indicate that they were of the hysterical kind. At the same time, it is unusual for a girl of ten years old to exhibit the symptoms of hysteria. We rarely find any manifestation of the hysterical condition, until after the appearance of the catamenia. She was persuaded to marry at the age

of fifteen, and, as she had no great affection for her husband, domestic squabbles occurred very soon after their union, and in some of these disagreements her husband used violence, and in consequence she had a return of the fits. At last they agreed to separate, but circumstances obliged them to have occasional interviews, and of necessity scenes of violence were repeated on each occasion. It was at one of these rencontres that the present attack commenced. The influence of moral causes in inducing the paroxysm is very manifest in this history. Ever since fifteen years of age, she stated, the slightest excitement would bring one on. She is evidently a woman of strong passions and violent temper, and possibly her partner was as much "sinned against as sinning." The same hysterical cast of features existed as in the case of Harriet B——, with which I commenced the lecture, and there were many of the same peculiarities of the hysterical constitution.

There is a curious family history in this case. Her paternal grandfather and paternal uncle had fits, and she was the mother of nine children, eight of whom died, seven of them in convulsions!

And now let me refer briefly to treatment. I would just remark here that the same general principles of treatment are applicable, whether the affection be chiefly manifested in a particular part of the body, as in the case of Harriet B——, or

whether it affects the system generally, as in the case of Amelia D——.

Before, however, we discuss the treatment, we must endeavour to form some notion of the nature of hysteria. What is this hysterical condition? To answer this question properly would, I fear, occupy me too long, but I may tell you what it is not, by which you may be guided in your practice, and be enabled to meet the disease by the proper treatment. Now, in the first place, hysteria does not result from inflammation. It is not a disease of an inflammatory type, and no part of the system whatever is in a state of inflammation. Hysteria, no doubt, occurs chiefly in persons who have a peculiar character of nervous system, very often inherited from nervous or gouty parents. It depends, partly upon this original conformation of nervous system, and partly upon a depraved state of general nutrition affecting the whole body, and accompanied by a morbid state of the blood. It is always very much influenced by a disordered condition of some of the great emunctories. Hence we must take care that the powers of the patient are not in any way diminished by the treatment to which she is subjected, or by the occurrence of fluxes from any part of the body which may tend to lower her strength. We must also be careful not to overlook any derangement of the digestive organs; we must uphold the strength, and endeavour to improve the patient's health

in every way that comes within her powers of digestion.

Although the hysterical condition is mainly physical, and, as such, transmissible from parent to offspring, you will not lose sight of the fact, that it is readily affected by moral influences. A sound education, as regulating the habits, is of primary importance to the hysterical patient, and a judiciously-directed moral treatment is essentially necessary, to give full effect to such physical remedies as may be applicable to the case. On the other hand, an ill-regulated mind and an imperfectly subjugated will are among the most frequent moral conditions which tend to excite and prolong the disease.

When you meet with a case, such as the present, in which a violent paroxysm has occurred, what are you to do? Are you to look on and do nothing? The friends will anxiously inquire if nothing can be done to relieve the patient. I confess that I cannot tell you of anything, which will, with certainty, quickly cut the paroxysm short, but at the same time certain means may be had recourse to, for the purpose of diminishing its violence and duration. Whatever tends to rouse the patient, without alarming, or creating exhaustion, is likely to prove useful, such as shaking her without violence, or throwing cold water in her face, or splashing her well with water. It is desirable to get the bowels to act freely as soon as possible.

Means of this kind were used in the case of Amelia D——, and an enema, containing half an ounce of spirits of turpentine with an ounce of tincture of assafœtida, was administered. She took, likewise, for a short time twenty minims of the fetid spirit of ammonia, and ten minims of tincture of hyoscyamus in camphor mixture at short intervals.

If the patient has been indulging too freely in beer or spirits, (as is often the case with persons brought into the hospital,) or if you have reason to suspect that any indigestible food has been taken, an emetic or the stomach-pump will be of great service. It rarely happens that taking blood, or other antiphlogistic treatment, is requisite.

The patient, Amelia D——, recovered in three or four hours from her fits, and slept the rest of the night. Next morning she was perfectly tranquil, and could not be prevailed on to remain in the hospital.

Catalepsy occurs in men, and, judging from what I have seen, in a form much more severe and fatal than in women. In men, as in the other sex, it is associated with exhausting causes and demoralizing influences, and is altogether a more serious malady, destructive to mind as well as body. At some future time I may have an opportunity of discussing the disease as it occurs in males.

THE END.

LONDON:
SAVILL AND EDWARDS, PRINTERS, CHANDOS STREET,
COVENT GARDEN.

BOUND BY
EDMONDS & REMNANTS
LONDON